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L2	L1 and (transgen$ or knockout or disrupt$)	29	L2
L1	melanocyte stimulating hormone receptor or MSH R	65	L1

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=> s melanocyte stimulating hormone receptor or MSH-R
L1 168 MELANOCYTE STIMULATING HORMONE RECEPTOR OR MSH-R

=> s l1 and (knockout or transgen? or disrupt?)
L2 3 L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT?)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 2 DUP REM L2 (1 DUPLICATE REMOVED)

=> d bib abs

L3 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2001:103127 BIOSIS
DN PREV200100103127
TI Enhancement of MSH receptor- and GAL4-mediated gene transfer by switching the nuclear import pathway.
AU Chan, C.-K.; Jans, D. A. (1)
CS (1) Nuclear Signalling Laboratory, Division of Biochemistry and Molecular Biology, John Curtin School of Medical Research, Canberra City, ACT 2601, Australia
SO Gene Therapy, (January, 2001) Vol. 8, No. 2, pp. 166-171. print. ISSN: 0969-7128.
DT Article
LA English
SL English
AB Efficient nuclear delivery of plasmid DNA represents a major barrier in nonviral gene transfer. One approach has been to use DNA-binding proteins such as GAL4 from yeast as DNA carriers with nuclear targeting properties. We recently showed, however, that GAL4 is inefficient in targeting DNA to the nucleus because its DNA-binding and nuclear targeting activities are mutually exclusive, which relates to the fact that GAL4 nuclear import occurs via a novel pathway. Here, we 'switch' this pathway to a more conventional one by adding a modified poly-lysine to which an optimized nuclear targeting signal, based on that of the SV40 large T-antigen, is linked. We also use a chimeric GAL4-alpha-melanocyte stimulating hormone (MSH) fusion protein to enable gene transfer to cells expressing the MSH receptor. Switching the nuclear import pathway of the transfecting complex significantly enhances receptor-mediated gene transfer through enabling interaction with desired components of the cellular nuclear import machinery. The present study represents the first demonstration that nuclear targeting signals can enhance receptor-mediated gene delivery, the approaches having important relevance to research and clinical applications, such as in generating ***transgenic*** or knock-out animals, or in gene therapy.

=> d bib abs 2

L3 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 1997:69526 BIOSIS
DN PREV199799368729
TI Role of melanocortinergic neurons in feeding and the agouti obesity syndrome.
AU Fan, Wei; Boston, Bruce A.; Kesterson, Robert A.; Hruby, Victor J.; Cone, Roger D. (1)
CS (1) Vollum Inst. Advanced Biomedical Res., Oregon Health Sci. Univ., 3181 S.W. Sam Jackson Park Rd., Portland, OR 97201, USA
SO Nature (London), (1997) Vol. 385, No. 6612, pp. 165-168. ISSN: 0028-0836.
DT Article
LA English
AB Dominant alleles at the agouti locus (A) cause an obesity syndrome in the mouse, as a consequence of ectopic expression of the agouti peptide. This peptide, normally only found in the skin, is a high-affinity antagonist of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1-R), thus explaining the inhibitory effect of agouti on eumelanin pigment synthesis. The agouti peptide is also an antagonist of the hypothalamic melanocortin-4 receptor (MC4-R). To test the hypothesis that agouti causes obesity by antagonism of hypothalamic melanocortin receptors, we identified cyclic melanocortin analogues that are potent agonists or antagonists of the neural MC3 (refs 11, 12) and MC4 receptors. Intracerebroventricular administration of the agonist, MTII, inhibited feeding in four models of hyperphagia: fasted C57BL/6J, ob/ob, and A-Y mice, and mice injected with neuropeptide Y. Co-administration of the specific melanocortin antagonist and agouti-mimetic SHU9119 completely blocked this inhibition. Furthermore, administration of SHU9119 significantly enhanced nocturnal feeding, or feeding stimulated by a prior fast. Our data show that melanocortinergic neurons exert a tonic inhibition of feeding behaviour. Chronic ***disruption*** of this inhibitory signal is a likely explanation of the agouti obesity syndrome.

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(FILE 'HOME' ENTERED AT 13:31:33 ON 11 MAR 2002)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 13:38:59 ON 11 MAR 2002
L1 168 S MELANOCYTE STIMULATING HORMONE RECEPTOR OR MSH-R
L2 3 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT?)
L3 2 DUP REM L2 (1 DUPLICATE REMOVED)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L4 108 DUP REM L1 (60 DUPLICATES REMOVED)

=> s l4 and py<2000
1 FILES SEARCHED
L5 94 L4 AND PY<2000

=> d bib abs 1-20

L5 ANSWER 1 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2002:45799 BIOSIS
DN PREV200200045799
TI DNA encoding alpha ***melanocyte*** ***stimulating*** ***hormone*** ***receptor***
AU Cone, R. D.; Mountjoy, K. G.
CS Oregon City, Oreg. USA
ASSIGNEE: STATE OF OREGON, ACTING BY AND THROUGH THE OREGON STATE BOARD OF
HIGHER EDUCATION ON BEHALF OF THE OREGON HEALTH SCIENCES UNIVERSITY, A
NON-PROFIT ORGANIZATION
PI US 5532347 July 2, 1996
SO Official Gazette of the United States Patent and Trademark Office Patents, (***July 2, 1996***) Vol. 1188, No. 1, pp. 464.
ISSN: 0098-1133
DT Patent
LA English

L5 ANSWER 2 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2000:89582 BIOSIS
DN PREV200000089582
TI The ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1-R) gene as a tool in evolutionary studies of Artiodactyles.
AU Klungland, Helge (1); Roed, Knut H.; Nesbo, Camilla L.; Jakobsen, Kjetill S.; Vage, Dag I.
CS (1) Department of Animal Science, Agricultural University of Norway, N-1432, As, Norway
SO Hereditas (Lund), (***Oct., 1999***) Vol. 131, No. 1, pp. 39-46.
ISSN: 0018-0661
DT Article
LA English
SL English
AB The complete coding region of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1-R) gene was characterized in species belonging to the two families Bovidae and Cervidae; cattle (Bos taurus), sheep (Ovis aries), goat (Capra hircus), muskox (Ovibos moschatus), roe deer (Capreolus capreolus), reindeer (Rangifer tarandus), moose (Alces alces), red deer (Cervus elaphus) and fallow deer (Dama dama). This well conserved gene is a central regulator of mammalian coat colour. Examination of the interspecies variability revealed a 5.3-6.8% divergence between the Cervidae and Bovidae families, whereas the divergence within the families were 1.0-3.1% and 1.2.-4.6%, respectively. Complete identity was found when two subspecies of reindeer, Eurasian tundra reindeer (R.t. tarandus) and Svalbard reindeer (R.t. platyrhynehus), were analyzed. An rooted phylogenetic tree based on Bovidae and Cervidae MC1-R DNA sequences was in complete agreement with current taxonomy, and was supported by bootstrapping analysis. Due to different frequencies of silent vs. replacement mutations, the amino acid based phylogenetic tree contains several dissimilarities when compared to the DNA based phylogenetic tree.

L5 ANSWER 3 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2000:84595 BIOSIS
DN PREV200000084595
TI Coat colour variation in cattle caused by mutations in the MC1-R and c-kit genes.
AU Klungland, Helge (1); Olsen, Hanne Gro (1); Vage, Dag Inge (1)
CS (1) Department of Animal Science, Agricultural University of Norway, 1432, As-NLH Norway
SO Pigment Cell Research, (1999) No. SUPPL. 7, pp. 67.
Meeting Info.: XVIIth International Pigment Cell Conference Nagoya, Japan October 30-November 3, 1999
ISSN: 0893-5785
DT Conference
LA English

L5 ANSWER 4 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2000:82205 BIOSIS
DN PREV200000082205
TI Spectrum of mutation at the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1R) The genetics of red hair in Caucasians.
AU Urquhart, Andy (1); Grimes, Eileen (1); Noake, Penelope (*); Dixon, Lindsay (1); Lowe, Alex (1)
CS (1) Forensic Science Service, Birmingham UK
SO Pigment Cell Research, (1999) No. SUPPL. 7, pp. 66.
Meeting Info.: XVIIth International Pigment Cell Conference Nagoya, Japan October 30-November 3, 1999
ISSN: 0893-5785
DT Conference
LA English

L5 ANSWER 5 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2000:75798 BIOSIS
DN PREV200000075798
TI Presence of the dominant extension allele ED in red and mosaic cattle.
AU Klungland, Helge (1); Vage, Dag Inge
CS (1) Department of Animal Science, Agricultural University of Norway, NLH N-1432, As, Norway
SO Pigment Cell Research, (***Dec., 1999***) Vol. 12, No. 6, pp. 391-393.
ISSN: 0893-5785
DT Article
LA English
SL English
AB The ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1-R) is a central regulator of mammalian coat colour, encoded by the extension locus. In cattle, the dominant extension allele ED is associated with the production of black pigment in coloured areas. Genotyping of the MC1-R gene in a bull with mosaic expression of red vs. black pigment verified the existence of the ED allele, in spite of the fact that the majority of the animal is red coloured. No further mutations were found within the ED variant of the MC1-R gene, which was inherited from a completely red mother (genotype ED/e).

L5 ANSWER 6 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1999:494936 BIOSIS
DN PREV199900494936
TI Cytochrome P450 CYP2D6 genotypes: Association with hair colour, Breslow thickness and ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** alleles in patients with malignant melanoma.
AU Strange, Richard C. (1); Ellison, Tracy; Ichii-Jones, Fumiyo; Bath, Joanna; Hoban, Paul; Lear, John T.; Smith, Andrew G.; Hutchinson, Peter E.; Osborne, Joy; Bowers, Bill; Jones, Peter W.; Fryer, Anthony A.
CS (1) School of Postgraduate Medicine, Keele University, North Staffordshire Hospital, Stoke-on-Trent, Staffordshire, ST4 7PA, UK
SO Pharmacogenetics, (***June, 1999***) Vol. 9, No. 3, pp. 269-276.
ISSN: 0960-314X.
DT Article
LA English
SL English
AB We previously identified associations between polymorphism in the cytochrome P450 CYP2D6 gene and outcome in several cancers. We have now examined the hypothesis that homozygosity for the mutant alleles, CYP2D6*4 and CYP2D6*3, is associated with susceptibility and outcome in malignant melanoma. Outcome was assessed by Breslow thickness. We first confirmed previous reports that these mutant alleles are associated with increased susceptibility to malignant melanoma. For example, the frequency of homozygosity for CYP2D6*4 was significantly greater (P = 0.006, chi-squared 1 d.f. = 7.4, odds ratio 2.2, 95% confidence interval 1.2, 3.9) in cases (9.1%) than in control individuals (4.3%). The frequency of homozygosity for the mutant alleles was next examined in the malignant melanoma cases grouped on the basis of characteristics associated with malignant melanoma risk. Homozygosity was significantly more common (P = 0.038) in cases with red/blonde hair than in those with brown/black hair. We found no associations between the CYP2D6 genotype and sex, skin type or eye colour. The possible association of CYP2D6 with outcome was assessed by comparing genotype frequencies in patients with tumours of Breslow thickness < 1.5 mm with those whose tumours were gtoreq 1.5 mm. In patients with red/blonde, but not brown or black hair, homozygosity for CYP2D6*4 was significantly associated with thicker lesions in a multivariate model (P = 0.036). We further examined the association of CYP2D6*4 homozygosity with red/blonde hair by classifying patients on the basis of homo- or heterozygosity for wild-type or val92met, asp294his or asp84glu ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1R) alleles. None of the nine patients with brown/black hair with the asp294his allele were homozygotes for CYP2D6*4. By contrast, in the patients with red/blonde hair, three of five cases with asp294his were homozygotes for the mutant CYP2D6 allele. The difference in the frequency of CYP2D6*4 homozygotes in the red/blonde cases with wild-type MC1R alleles compared with those with asp294his was significant (exact P = 0.029). No associations between val92his or asp84glu and CYP2D6 alleles were identified.

L5 ANSWER 7 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1999:366170 BIOSIS
DN PREV199900366170
TI Association of NAD(P)H quinone oxidoreductase (NQO1) null with numbers of basal cell carcinomas. Use of a multivariate model to rank the relative importance of this polymorphism and those at other relevant loci.
AU Clairmont, Annette; Sies, Helmut; Ramachandran, Sudarshan; Lear, John T.; Smith, Andrew G.; Bowers, Bill; Jones, Peter W.; Fryer, Anthony A.; Strange, Richard C. (1)
CS (1) Clinical Biochemistry Research Laboratory, School of Postgraduate Medicine, Centre for Cell and Molecular Medicine, Keele University, North Staffordshire Hospital, Hartshill, Stoke-on-Trent, Staffordshire, ST4 7QB, UK
SO Carcinogenesis (Oxford), (***July, 1999***) Vol. 20, No. 7, pp. 1235-1240.
ISSN: 0143-3334
DT Article
LA English
SL English
AB Glutathione S-transferase GSTM1 B and GSTT1 null, and cytochrome P450 CYP2D6 EM have been associated with cutaneous basal cell carcinoma (BCC)

numbers, although their quantitative effects show that predisposition to many BCC is determined by an unknown number of further loci. We speculate that other loci that determine response to oxidative stress, such as NAD(H) quinone oxidoreductase (NQO1) are candidates. Accordingly, we assessed the association between NQO1 null and BCC numbers primarily to rank NQO1 null in a model that included genotypes already associated with BCC numbers. We found that only 14 out of 457 cases (3.1%) were NQO1 null. This frequency did not increase in cases with characteristics linked with BCC numbers including gender, skin type, a truncal lesion or more than one new BCC at any presentation (MPP). However, the mean number of BCC in NQO1*0 homozygotes was greater than in wild-type allele homozygotes and heterozygotes, although the difference was not quite significant (P = 0.06). These data reflect the link between NQO1 null and BCC numbers in the 42 MPP cases rather than the whole case group. We identified an interaction between NQO1 null and GSTT1 null that was associated with more BCC (P = 0.04), although only four cases had this combination. The relative influence of NQO1 null was studied in a multivariate model that included (i) 241 patients in whom GSTM1 B, GSTT1 null and CYP2D6 EM genotype data were available, and (ii) 101 patients in whom these genotypes, as well as data on GSTM3, CYP1A1 and ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1R) genotypes were available. NQO1 null (P = 0.001) and MC1R asp294/asp294 (P = 0.03) were linked with BCC numbers, and the association with CYP2D6 EM approached significance (P = 0.08). In a stepwise regression model only these genotypes were significantly associated with BCC numbers with NQO1 null being the most powerful predictor.

L5 ANSWER 8 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:343142 BIOSIS
DN PREV199900343142
TI alpha-MSH and its receptors in regulation of tumor necrosis factor-alpha production by human monocyte/macrophages.
AU Taherzadeh, S.; Sharma, S.; Chhajlani, V.; Gantz, I.; Rajora, N.; Demitri, M. T.; Kelly, L.; Zhao, H.; Ichiyama, T.; Catania, A.; Lipton, J. M. (1)
CS (1) Univ. of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., Dallas, TX, 75235-9040 USA
SO American Journal of Physiology, (***May, 1999***) Vol. 276, No. 5 PART 2, pp. R1289-R1294.
ISSN: 0002-9513.
DT Article
LA English
SL English
AB The hypothesis that macrophages contain an autocrine circuit based on melanocortin (ACTH and alpha-melanocyte-stimulating hormone (alpha-MSH)) peptides has major implications for neuroimmunomodulation research and inflammation therapy. To test this hypothesis, cells of the THP-1 human monocyte/macrophage line were stimulated with lipopolysaccharide (LPS) in the presence and absence of alpha-MSH. The inflammatory cytokine tumor necrosis factor (TNF)-alpha was inhibited in relation to alpha-MSH concentration. Similar inhibitory effects on TNF-alpha were observed with ACTH peptides that contain the alpha-MSH amino acid sequence and act on melanocortin receptors. Nuclease protection assays indicated that expression of the human melanocortin-1 receptor subtype (hMC-1R) occurs in THP-1 cells; Southern blots of RT-PCR product revealed that additional subtypes, hMC-3R and hMC-5R, also occur. Incubation of resting macrophages with antibody to hMC-1R increased TNF-alpha concentration; the antibody also markedly reduced the inhibitory influence of alpha-MSH on TNF-alpha in macrophages treated with LPS. These results in cells known to produce alpha-MSH at rest and to increase secretion of the peptide when challenged are consistent with an endogenous regulatory circuit based on melanocortin peptides and their receptors. Targeting of this neuroimmunomodulatory circuit in inflammatory diseases in which myelomonocytic cells are prominent should be beneficial.

L5 ANSWER 9 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:293085 BIOSIS
DN PREV199900293085
TI The ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** polymorphism. Association of the V92M and A294H alleles with basal cell carcinoma.
AU Jones, Fumiyo Ichii, Ramachandran, Sudarshan, Lear, John, Smith, Andrew, Bowers, Bill, Ollier, William E. R., Jones, Peter, Fryer, Anthony A., Strange, Richard C. (1)
CS (1) Clinical Biochemistry Research Group, School of Postgraduate Medicine, North Staffordshire Hospital, Keele University, Stoke-on-Trent, Staffordshire UK
SO Clinica Chimica Acta, (***April, 1999***) Vol. 282, No. 1-2, pp. 125-134.
ISSN: 0009-8981.
DT Article
LA English
SL English
AB Allelic variants in the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1R) gene are susceptibility/outcome candidates for cutaneous basal cell carcinoma (BCC). We identified the val92met (V92M) and asp294his (A294H) alleles in 311 cases and 190 controls. The cases included four homo- and 53 heterozygotes for V92M and 12 heterozygotes for A294H and two compound heterozygotes (V92M/A294H). Allele frequencies were similar in controls. In the cases, we found no association between the alleles and skin type though A294H was more common in those with red hair (4/19) than with other hair colours (6/163) (P = 0.012). V92M was not associated with BCC numbers. Cases with A294H had fewer BCC in comparison with those without the allele though the difference was not significant. After inclusion of red hair in the model, A294H was significantly associated with fewer tumours. While MC1R alleles are attractive candidates for BCC, the variants studied did not influence susceptibility. The association with outcome was relatively weak. The large number of MC1R alleles and their low frequency, make assessment of the importance of this gene in the pathogenesis of skin cancers difficult.

L5 ANSWER 10 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:137364 BIOSIS
DN PREV199900137364
TI Presence of the dominant extension allele ED in red and mosaic cattle.
AU Klungland, H.; Vage, D. I.
CS Dep. Anim. Sci., Agric. Univ. Norway, PO Box 5025, N-1432 As Norway
SO Animal Genetics, (***Dec., 1998***) Vol. 29, No. SUPPL. 1, pp. 50.
Meeting Info.: 26th International Conference on Animal Genetics Auckland, New Zealand August 9-14, 1998
ISSN: 0268-9146.
DT Conference
LA English

L5 ANSWER 11 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:135343 BIOSIS
DN PREV199900135343
TI Molecular characterization and genetic analysis of black coat colour in the horse.
AU Lieto, L. D. (1); Cothran, E. G. (1); Sponenberg, D. P.
CS (1) Univ. Kentucky, Lexington, KY USA
SO Animal Genetics, (***Dec., 1998***) Vol. 29, No. SUPPL. 1, pp. 57.
Meeting Info.: 26th International Conference on Animal Genetics Auckland, New Zealand August 9-14, 1998
ISSN: 0268-9146.
DT Conference
LA English

L5 ANSWER 12 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:98298 BIOSIS
DN PREV199900098298
TI Molecular and pharmacological characterization of dominant black coat color in sheep.
AU Vage, Dag Inge (1); Klungland, Helge; Lu, Dongsi; Cone, Roger D.
CS (1) Dep. Anim. Sci., Agric. Univ., Norway, P.O. Box 5025, N-1432 As Norway
SO Mammalian Genome, (***Jan., 1999***) Vol. 10, No. 1, pp. 39-43.
ISSN: 0938-8990.
DT Article
LA English
AB Dominant black coat color in sheep is predicted to be caused by an allele ED at the extension locus. Recent studies have shown that this gene encodes the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1-R). In mouse and fox, naturally occurring mutations in the coding region of MC1-R produce a constitutively activated receptor that switches the synthesis from phaeomelanin to eumelanin within the melanocyte, explaining the black coat color observed phenotypically. In the sheep, we have identified a MetfwdarwLys mutation in position 73 (M73K) together with a Asp fwdarw Asn change at position 121 (D121N) showing complete cosegregation with dominant black coat color in a family lineage. Only the M73K mutation showed constitutive activation when introduced into the corresponding mouse receptor (mMC1-R) for pharmacological analysis; however, the position corresponding to D121 in the mouse receptor is required for high affinity ligand binding. The pharmacological profile of the M73K change is unique compared to the constitutively active E92K mutation in the sombre mouse and C123R mutation in the Alaska silver fox, indicating that the M73K change activates the receptor via a mechanism distinct from these previously characterized mutations.

L5 ANSWER 13 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:88552 BIOSIS
DN PREV199900088552
TI Human pigmentation genes and their response to solar UV radiation.
AU Sturm, Richard A. (1)
CS (1) Cent. Mol. Cell Biol., Univ. Queensl., Brisbane, Qld 4072 Australia
SO Mutation Research, (***Nov. 9, 1998***) Vol. 422, No. 1, pp. 69-76.
ISSN: 0027-5107.
DT General Review
LA English
AB Identification and characterisation of the genes involved in melanin pigment formation, together with the study of how their action is influenced by exposure to UV radiation, is providing a molecular understanding of the process of skin photoprotection through tanning. The mechanisms underlying this change in epidermal melanin involve both a transcriptional response of the pigmentation genes and post-translational control of the melanin biosynthetic pathway. UV rays are known to interact with numerous molecules within cells, and among these the photochemical reactions involving lipids and DNA are implicated in modulating melanogenesis. The combination of DNA damage, the formation of diacylglycerol, and the action of the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** are all likely to

be involved in UV-induced tanning

L5 ANSWER 14 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1999 72088 BIOSIS
DN PREV199900072088
TI Alpha- ***Melanocyte*** ***stimulating*** ***hormone*** ***receptor***
AU Cone, R. D.; Mountjoy, K. G.
CS Oregon City, Oreg. USA
ASSIGNEE: OREGON HEALTH SCIENCES UNIVERSITY
PI US 5849871 Dec. 15, 1998
SO Official Gazette of the United States Patent and Trademark Office Patents, (***Dec. 15, 1998***) Vol. 1217, No. 3, pp. 2637.
ISSN: 0098-1133
DT Patent
LA English

L5 ANSWER 15 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1999.6018 BIOSIS
DN PREV199900006018
TI Design and characterization of alpha-melanotropin peptide analogs cyclized through rhenium and technetium metal coordination.
AU Giblin, Michael F.; Wang, Nannan; Hoffman, Timothy J.; Jurisson, Silvia S.; Quinn, Thomas P. (1)
CS (1) Dep. Biochemistry, 117 Schweitzer Hall, Univ. Missouri, Columbia, MO 65211 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (***Oct. 27, 1998***) Vol. 95, No. 22, pp. 12814-12818.
ISSN: 0027-8424
DT Article
LA English
AB alpha-Melanocyte stimulating hormone (alphaMSH) analogs, cyclized through site-specific rhenium (Re) and technetium (Tc) metal coordination, were structurally characterized and analyzed for their abilities to bind alpha-MSH receptors present on melanoma cells and in tumor-bearing mice. Results from receptor-binding assays conducted with B16-F1 murine melanoma cells indicated that receptor-binding affinity was reduced to approximately 1% of its original levels after Re incorporation into the cyclic CyS4,10, D-Phe7-alpha-MSH4-13 analog. Structural analysis of the Re-peptide complex showed that the disulfide bond of the original peptide was replaced by thiolate-metal-thiolate cyclization. A comparison of the metal-bound and metal-free structures indicated that metal complexation dramatically altered the structure of the receptor-binding core sequence. Redesign of the metal binding site resulted in a second-generation Re-peptide complex (ReCCMSH) that displayed a receptor-binding affinity of 2.9 nM, 25-fold higher than the initial Re-alpha-MSH analog. Characterization of the second-generation Re-peptide complex indicated that the peptide was still cyclized through Re coordination, but the structure of the receptor-binding sequence was no longer constrained. The corresponding 99mTc- and 188ReCCMSH complexes were synthesized and shown to be stable in phosphate-buffered saline and to challenges from diethylenetriaminepentaacetic acid (DTPA) and free cysteine. In vivo, the 99mTcCCMSH complex exhibited significant tumor uptake and retention and was effective in imaging melanoma in a murine-tumor model system. Cyclization of alpha-MSH analogs via 99mTc and 188Re yields chemically stable and biologically active molecules with potential melanoma-imaging and therapeutic properties.

L5 ANSWER 16 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1998:497866 BIOSIS
DN PREV199800497866
TI Mahogany (mg) stimulates feeding and increases basal metabolic rate independent of its suppression of agouti.
AU Dinulescu, Daniela M.; Fan, Wei; Boston, Bruce A.; McCall, Kathleen; Lamoreux, M. Lynn; Moore, Karen J.; Montagno, Jill; Cone, Roger D. (1)
CS (1) Vollum Inst., Oregon Health Sci., Univ., 3181 SW Sam Jackson Park Rd., Portland, OR 97201 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (***Oct. 13, 1998***) Vol. 95, No. 21, pp. 12707-12712.
ISSN: 0027-8424.
DT Article
LA English
AB The mahogany (mg) locus originally was identified as a recessive suppressor of agouti, a locus encoding a skin peptide that modifies coat color by antagonizing the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** or MC1-R. Certain dominant alleles of agouti cause an obesity syndrome when ectopic expression of the peptide aberrantly antagonizes the MC4-R, a related ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** expressed in hypothalamic circuitry and involved in the regulation of feeding behavior and metabolism. Recent work has demonstrated that mg, when homozygous, blocks not only the ability of agouti to induce a yellow coat color when expressed in the skin of the lethal yellow mouse (AY), but also the obesity resulting from ectopic expression of agouti in the brain. Detailed analysis of mg/mg AYa animals, presented here, demonstrates that mg/mg blocks the obesity, hyperinsulinemia, and increased linear growth induced by ectopic expression of the agouti peptide. Remarkably, however, mg/mg did not reduce hyperphagia in the AY/a mouse. Furthermore, mg/mg induced hyperphagia and an increase in basal metabolic rate in the C57BL/6J mouse in the absence of AY. Consequently, although mahogany is broadly required for agouti peptide action, it also appears to be involved in the control of metabolic rate and feeding behavior independent of its suppression of agouti.

L5 ANSWER 17 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1998.434529 BIOSIS
DN PREV199800434529
TI Modulation of ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** expression on normal human melanocytes: Evidence for a regulatory role of ultraviolet B, interleukin-1alpha, interleukin-1beta, endothelin-1 and tumour necrosis factor-alpha.
AU Funasaka, Y. (1); Chakraborty, A. K.; Hayashi, Y.; Komoto, M.; Ohashi, A.; Nagahama, M.; Inoue, Y.; Pawelek, J.; Ichihashi, M.
CS (1) Dep. Dermatol., Kobe Univ. Sch. Med., 5-1 Kusunoki-cho 7-chome, Chu-ku, Kobe 650 Japan
SO British Journal of Dermatology, (***Aug. 1998***) Vol. 139, No. 2, pp. 216-224.
ISSN: 0007-0963
DT Article
LA English
AB Melanocyte-stimulating hormone (MSH) receptor binding activity and melanocortin-1 receptor (MC1-R) gene expression on normal human melanocytes have been studied as responses to the effects of ultraviolet B (UVB), interleukin-1 (IL-1), endothelin-1 (ET-1) and tumour necrosis factor-alpha (TNF-alpha), which are known as UV sensitive regulators of melanocytic function. MSH receptor (***MSH*** - ***R***) binding activity was upregulated by UVB, IL-1alpha, -1beta and ET-1, but was downregulated by TNF-alpha. Northern blot analysis showed that MC1-R mRNA expression was induced 24h after UVB irradiation in a dosedependent manner, and that 24-h treatment with ET-1 also induced an expression of MC1-R mRNA, whereas TNF-alpha downregulated the expression. In addition, IL-1alpha and -1beta have a small but real inductive effect on MC1-R mRNA expression. Taken together, our results suggest a model in which higher MC1-R mRNA expression is accompanied by upregulation of ***MSH*** - ***R*** binding activity, and enhanced by UVB or cytokines sensitive to UVB. Such a regulatory system would enable normal human melanocytes to respond to MSH more efficiently and induce an increase of melanization of the skin through the MSH/ ***MSH*** - ***R*** system after UVB radiation.

L5 ANSWER 18 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1998.405474 BIOSIS
DN PREV199800405474
TI Susceptibility to melanoma: Influence of skin type and polymorphism in the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** gene.
AU Ichii-Jones, Fumiyo; Lear, John T.; Heagerty, Adrian H. M.; Smith, Andrew G.; Hutchinson, Peter E.; Osborne, Joy; Bowers, Bill; Jones, Peter W.; Davies, Eric; Oliver, William E. R.; Thomson, Wendy; Yengi, Lillian; Bath, Joanna; Fryer, Anthony A.; Strange, Richard C. (1)
CS (1) Sch. Postgraduate Med., Univ. Keele, North Staffordshire Hosp., Stoke-on-Trent, Staffordshire ST4 7PA UK
SO Journal of Investigative Dermatology, (***Aug., 1998***) Vol. 111, No. 2, pp. 218-221.
ISSN: 0022-202X
DT Article
LA English
AB Allelic variation at the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1R) gene has been linked with sun-sensitive skin types, suggesting it is a susceptibility candidate for melanoma. We determined the frequency of the val92met, asp294his, and asp84glu MC1R alleles in 190 Caucasian controls and 306 melanoma cases and studied their association with skin type and hair color. The percentage of controls with at least one val92met, asp294his, or asp84glu allele was 17.3%, 6.8%, and 3.5%, respectively. Individually, frequencies of the val92met, asp294his, or asp84glu alleles in the controls with skin types 3 and 4 were similar to those with skin types 1 and 2. Trend analysis, however, did identify an association (exact p = 0.048, two-sided test) between skin type and MC1R variants in the group comprising all controls with any one or more of these alleles. There was no association between MC1R alleles and hair color. Allele frequencies were not different in melanoma cases and controls. There were no associations between skin types and the proportion of cases with the asp294his or asp84glu alleles, though the association between skin type and the val92met allele approached significance (exact p = 0.09, two-sided test). Unexpectedly, in the group comprising all cases with one or more variant alleles, the proportion of subjects with variant alleles increased with skin types associated with tanning rather than burning, although trend analysis showed that this association did not quite reach statistical significance (exact p = 0.08, two-sided test). Asp84glu (but not val92met or asp294his) variant alleles were more common in subjects with blonde hair, although the relationship between the asp84glu allele and hair color did not achieve statistical significance (chi32 = 6.16, exact p = 0.10). We interpret the data presented as indicating that polymorphism at MC1R does not appear a major determinant of skin type, at least in terms of these allelic variants. Furthermore, considered alone, these alleles are not susceptibility candidates for malignant melanoma.

L5 ANSWER 19 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1998 392760 BIOSIS
DN PREV199800392760
TI Endothelin-1 is a paracrine growth factor that modulates melanogenesis of human melanocytes and participates in their responses to ultraviolet radiation
AU Tada, Akihiro (1); Suzuki, Itaru; Im, Sungbin; David, Mary Beth; Cornelius, James; Babcock, George; Nordlund, James J.; Abdel-Malek, Zalfa A.
CS (1) Dep. Dermatol., Univ. Cincinnati, P.O. Box 670592, Cincinnati, OH 45267-0592 USA
SO Cell Growth & Differentiation, (***July, 1998***) Vol. 9, No. 7, pp. 575-584.
ISSN: 1044-9523.
DT Article
LA English
AB Endothelin (ET)-1, alpha-melanocyte stimulating hormone (alpha-melanotropin, alpha-MSH), and basic fibroblast growth factor (bFGF) are keratinocyte-derived factors that interact synergistically to stimulate human melanocyte proliferation. ET-1 has a dose-dependent mitogenic effect on human melanocytes and a biphasic effect on melanogenesis: a stimulatory effect at subnanomolar concentrations, and an inhibitory effect at concentrations equal to or higher than 1 nM. Human melanocytes express ET B receptors. Brief treatment of melanocytes with ET-1 caused up-regulation of alphaMSH receptor mRNA but did not alter ET B receptor mRNA level. ET-1 modulates the response of human melanocytes to UV rays (UVRs). Treatment of melanocytes with 10 nM ET-1 immediately after exposure to UVRs enabled them to overcome the G, growth arrest. However, ET-1 did not inhibit p53 accumulation or p21Waf-1/SDI-1/Cip-1 overexpression, nor did it reverse the hypophosphorylated state of pRb or the reduction in Bc12 level in irradiated melanocytes. These results substantiate the role of ET-1 as a paracrine regulator that modulates the response of human melanocytes to UVRs.

L5 ANSWER 20 OF 94 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1998 376047 BIOSIS
DN PREV199800376047
TI Malignant melanoma skin type and polymorphism in the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** gene.
AU Lear, J. (1); Jones, F.; Smith, A.; Hutchinson, P.; Osbourne, J.; Ollier, W.; Fryer, A.; Strange, R.C.
CS (1) Dep. Dermatol., Bristol Royal Infirmary, Bristol UK
SO British Journal of Dermatology, (***April, 1998***) Vol. 138, No. 4, pp. 746.
Meeting Info.: Annual Meeting of the British Society for Investigative Dermatology and British Photodermatology Group Liverpool, England, UK April 22-24, 1998 British Photodermatology Group.
ISSN: 0007-0963.
DT Conference
LA English

=> d his

(FILE 'HOME' ENTERED AT 13:31:33 ON 11 MAR 2002)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 13:38:59 ON 11 MAR 2002
L1 168 S MELANOCYTE STIMULATING HORMONE RECEPTOR OR MSH-R
L2 3 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT?)
L3 2 DUP REM L2 (1 DUPLICATE REMOVED)
L4 108 DUP REM L1 (60 DUPLICATES REMOVED)
L5 94 S L4 AND PY<2000

=> s l1 and (brain or neuro?)
L6 22 L1 AND (BRAIN OR NEURO?)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 16 DUP REM L6 (6 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 16 ANSWERS - CONTINUE? Y/(N) y

L7 ANSWER 1 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE
1
AN 2001 79102 BIOSIS
DN PREV200100079102
TI Expression of the melanin-concentrating hormone receptor in porcine and human ciliary epithelial cells.
AU Hintermann, Edith; Erb, Carl; Talke-Messerer, Christiane; Liu, Rong; Tanner, Heidi; Flammer, Josef; Eberle, Alex N. (1)
CS (1) Department of Research (ZLF), University Hospital, Hebelstrasse 20, CH-4031, Basel: alex-n.eberle@unibas.ch Switzerland
SO IOVS, (January, 2001) Vol. 42, No. 1, pp. 206-209. print
DT Article
LA English
SL English
AB Purpose: To evaluate whether the receptors for melanin-concentrating hormone (MCH) and its functional antagonist alpha-melanocyte-stimulating hormone (alpha-MSH) are expressed in the ciliary epithelium. Furthermore, to examine whether MCH, a ***neuropeptide*** involved in fluid and electrolyte homeostasis, may influence ion flux mediated by Na,K (adenosine triphosphatase)-ATPase in a ciliary epithelial cell line. Methods: Expression of MCH receptors (MCH-R) and alpha-MSH receptors (***MSH*** - ***R***) on primary porcine ciliary pigmented epithelial (PE) cells and on a human nonpigmented ciliary epithelial (NPE) cell line, ODM-2 was investigated by radioligand binding studies and reverse transcription-polymerase chain reaction (RT-PCR). The MCH-R was further characterized by photocrosslinking. Influence of MCH on Na,K-ATPase activity was evaluated by an Rb+ transport assay. Results: MCH-R expression was observed at both the mRNA and protein levels in PE and NPE cells. In contrast, MSH-Rs were not detectable. At the mRNA level, expression of slc-1 was shown and with crosslinking, a 44-kDa protein was labeled. MCH showed no effect on Na,K-ATPase activity of NPE cells. Conclusions: The presence of MCH-R in ciliary epithelial cells of both human and porcine origin but the absence of MSH-Rs indicates that in these cells, MCH and alpha-MSH do not form a functionally antagonistic hormonal pair as they do in several other systems. Although effects of MCH on intestinal water and ion transport have been documented, a direct control of Na,K-ATPase activity was not detected in human NPE cells in vitro.

L7 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1999:589640 CAPLUS
DN 131:309750
TI Receptor-mediated modulation of murine mast cell function by .alpha.-melanocyte stimulating hormone
AU Adachi, Shiro; Nakano, Teruaki; Vliagoftis, Harrisios; Metcalfe, Dean D.
CS Laboratory of Allergic Disease, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD, 20892, USA
SO J. Immunol. (1999), 163(6), 3363-3368
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB The proopiomelanocortin (POMC)-derived ***neuropeptide*** .alpha.-MSH is known to modulate some aspects of inflammation through direct effects on T cells, B cells, and monocytes. To det. whether .alpha.-MSH might similarly influence mast cell responsiveness, mast cells were examd. to see if they expressed the receptor for .alpha.-MSH, melanocortin-1 (MC-1), and whether .alpha.-MSH altered mast cell function. The authors thus first identified MC-1 on bone marrow cultured murine mast cells (BMCMC) and a murine mast cell line (MCP-5) employing flow cytometry and through detection of specific binding. Subsequent treatment of mast cells with .alpha.-MSH increased the cAMP concn. in a characteristic biphasic pattern, demonstrating that .alpha.-MSH could affect intracellular processes. The authors next examd. the effect of .alpha.-MSH on mediator release and cytokine expression. IgE/DNP-human serum albumin-stimulated histamine release from mast cells was inhibited by .apprx.60% in the presence of .alpha.-MSH. Although activation of BMCMC induced the expression of mRNAs for the inflammatory cytokines IL-1 beta., IL-4, IL-6, TNF-.alpha., and the chemokine lymphotactin, mRNAs for IL-1 beta., TNF-.alpha., and lymphotactin were down-modulated in the presence of .alpha.-MSH. Finally, IL-3-dependent proliferative activity of BMCMC was slightly but significantly augmented by .alpha.-MSH. Thus, .alpha.-MSH may exert an inhibitory effect on the mast cell-dependent component of a specific inflammatory response.
RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1999 343142 BIOSIS
DN PREV199900343142
TI alpha-MSH and its receptors in regulation of tumor necrosis factor-alpha production by human monocyte/macrophages.
AU Taherzadeh, S.; Sharma, S.; Chhajlani, V.; Gantz, I.; Rajora, N.; Demitri, M.T.; Kelly, L.; Zhao, H.; Ichiyama, T.; Catania, A.; Lipton, J.M. (1)
CS (1) Univ. of Texas Southwestern Medical Center, 5323 Harry Hines Blvd., Dallas, TX, 75235-9040 USA
SO American Journal of Physiology, (May, 1999) Vol. 276, No. 5 PART 2, pp. R1289-R1294.
ISSN: 0002-9513.
DT Article
LA English
SL English
AB The hypothesis that macrophages contain an autocrine circuit based on melanocortin (ACTH and alpha-melanocyte-stimulating hormone (alpha-MSH)) peptides has major implications for ***neuroimmunomodulation*** research and inflammation therapy. To test this hypothesis, cells of the THP-1 human monocyte/macrophage line were stimulated with lipopolysaccharide (LPS) in the presence and absence of alpha-MSH. The inflammatory cytokine tumor necrosis factor (TNF)-alpha was inhibited in relation to alpha-MSH concentration. Similar inhibitory effects on TNF-alpha were observed with ACTH peptides that contain the alpha-MSH amino acid sequence and act on melanocortin receptors. Nuclease protection assays indicated that expression of the human melanocortin-1 receptor subtype (hMC-1R) occurs in THP-1 cells. Southern blots of RT-PCR product revealed that additional subtypes, hMC-3R and hMC-5R, also occur. Incubation of resting macrophages with antibody to hMC-1R increased TNF-alpha concentration, the antibody also markedly reduced the inhibitory

influence of alpha-MSH on TNF-alpha in macrophages treated with LPS. These results in cells known to produce alpha-MSH at rest and to increase secretion of the peptide when challenged are consistent with an endogenous regulatory circuit based on melanocortin peptides and their receptors. Targeting of this ***neuroimmunomodulatory*** circuit in inflammatory diseases in which myelomonocytic cells are prominent should be beneficial.

L7 ANSWER 4 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 2
AN 1998:497866 BIOSIS
DN PREV199800497866
TI Mahogany (mg) stimulates feeding and increases basal metabolic rate independent of its suppression of agouti.
AU Dinulescu, Daniela M.; Fan, Wei; Boston, Bruce A.; McCall, Kathleen; Lamoreux, M. Lynn; Moore, Karen J.; Montagno, Jill; Cone, Roger D. (1)
CS (1) Vollum Inst., Oregon Health Sci. Univ., 3181 SW Sam Jackson Park Rd., Portland, OR 97201 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (Oct. 13, 1998) Vol. 95, No. 21, pp. 12707-12712. ISSN: 0027-8424.
DT Article
LA English
AB The mahogany (mg) locus originally was identified as a recessive suppressor of agouti, a locus encoding a skin peptide that modifies coat color by antagonizing the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** or MC1-R. Certain dominant alleles of agouti cause an obesity syndrome when ectopic expression of the peptide aberrantly antagonizes the MC4-R, a related ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** expressed in hypothalamic circuitry and involved in the regulation of feeding behavior and metabolism. Recent work has demonstrated that mg, when homozygous, blocks not only the ability of agouti to induce a yellow coat color when expressed in the skin of the lethal yellow mouse (AY), but also the obesity resulting from ectopic expression of agouti in the ***brain***. Detailed analysis of mg/mg AYa animals, presented here, demonstrates that mg/mg blocks the obesity, hyperinsulinemia, and increased linear growth induced by ectopic expression of the agouti peptide. Remarkably, however, mg/mg did not reduce hyperphagia in the AY/a mouse. Furthermore, mg/mg induced hyperphagia and an increase in basal metabolic rate in the C57BL/6J mouse in the absence of AY. Consequently, although mahogany is broadly required for agouti peptide action, it also appears to be involved in the control of metabolic rate and feeding behavior independent of its suppression of agouti.

L7 ANSWER 5 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1997:489360 BIOSIS
DN PREV199799788563
TI Synthetic peptides derived from the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** MC1R can stimulate HLA-A2-restricted cytotoxic T lymphocytes that recognize naturally processed peptides on human melanoma cells.
AU Salazar-Onfray, Flavio; Nakazawa, Tsutomu; Chhajlani, Vijay; Petersson, Max; Karre, Klas; Masucci, Giuseppe; Celis, Esteban; Sette, Alessandro; Southwood, Scott; Appella, Ettore; Kiessling, Rolf (1)
CS (1) Microbiol. Tumor Biol. Cent., Karolinska Inst., S-171 77 Stockholm Sweden
SO Cancer Research, (1997) Vol. 57, No. 19, pp. 4348-4355. ISSN: 0008-5472.
DT Article
LA English
AB Human melanoma-specific HLA-A2 restricted CTLs have recently been shown to recognize antigens expressed by melanoma lines and normal melanocytes, including Melan-A/Mart-1, gp100, gp75, and tyrosinase. Herein, we define HLA-A2-restricted CTL epitopes from a recently cloned melanocortin 1 receptor (MCIR), which belongs to a new subfamily of the G-protein-coupled receptors expressed on melanomas and melanocytes. Thirty-one MCIR-derived peptides were selected on the basis of HLA-A2-specific motifs and tested for their HLA-A2 binding capacity. Of a group of 12 high or intermediate HLA-A2 binding peptides, three nonamers, MCIR244 (TiLLGIFFL), MC1R283 (FLALIICNA), and MCIR291 (AIIDPLIYA), were found to induce peptide-specific CTLs from peripheral blood mononuclear cells of healthy HLA-A2+ donors after repeated in vitro stimulation with peptide-pulsed antigen-presenting cells. The CTLs raised against these three HLA-A2+-restricted peptides could recognize naturally processed peptides from HLA-A2+ melanomas and from Cos7 cells cotransfected with MCIR and HLA-A2. CTLs induced by the MCIR291 peptide (but not induced or induced only to a very low extent by the other two MCR1 peptide epitopes) showed cross-reactions with two other members of the melanocortin receptor family, which are more broadly expressed on other tissues. Taken together, our findings have implications in relation both to autoimmunity and immunotherapy of malignant melanomas.

L7 ANSWER 6 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 3
AN 1997:69526 BIOSIS
DN PREV199799368729
TI Role of melanocortinergic ***neurons*** in feeding and the agouti obesity syndrome.
AU Fan, Wei; Boston, Bruce A.; Kesterson, Robert A.; Hruby, Victor J.; Cone, Roger D. (1)
CS (1) Vollum Inst. Advanced Biomedical Res., Oregon Health Sci. Univ., 3181 S.W. Sam Jackson Park Rd., Portland, OR 97201 USA
SO Nature (London), (1997) Vol. 385, No. 6612, pp. 165-168. ISSN: 0028-0836.
DT Article
LA English
AB Dominant alleles at the agouti locus (A) cause an obesity syndrome in the mouse, as a consequence of ectopic expression of the agouti peptide. This peptide, normally only found in the skin, is a high-affinity antagonist of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1-R), thus explaining the inhibitory effect of agouti on eumelanin pigment synthesis. The agouti peptide is also an antagonist of the hypothalamic melanocortin-4 receptor (MC4-R). To test the hypothesis that agouti causes obesity by antagonism of hypothalamic melanocortin receptors, we identified cyclic melanocortin analogues that are potent agonists or antagonists of the neural MC3 (refs 11, 12) and MC4 receptors. Intracerebroventricular administration of the agonist, MTII, inhibited feeding in four models of hyperphagia: fasted C57BL/6J, ob/ob, and A-Y mice, and mice injected with ***neuropeptide*** Y. Co-administration of the specific melanocortin antagonist and agouti-mimetic SHU9119 completely blocked this inhibition. Furthermore, administration of SHU9119 significantly enhanced nocturnal feeding, or feeding stimulated by a prior fast. Our data show that melanocortinergic ***neurons*** exert a tonic inhibition of feeding behaviour. Chronic disruption of this inhibitory signal is a likely explanation of the agouti obesity syndrome.

L7 ANSWER 7 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 4
AN 1995:110714 BIOSIS
DN PREV199598125014
TI Transcriptional induction of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** in ***brain*** metastases of murine K-1735 melanoma.
AU Radinsky, Robert (1); Beltran, Pedro J.; Tsan, Rachel; Zhang, Ruodan; Cone, Roger D.; Fidler, Isaiah J.
CS (1) Dep. Cell Biol., Box 173, Univ. Texas M.D. Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030 USA
SO Cancer Research, (1995) Vol. 55, No. 1, pp. 141-148. ISSN: 0008-5472.
DT Article
LA English
AB Metastatic K-1735 murine melanoma cells are amelanotic in culture or in the subcutis of syngeneic mice. When injected into the internal carotid artery, these cells produce melanotic ***brain*** metastases. The production of melanin in tumor cells growing in the ***brain*** was directly correlated with induction of ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (***MSH*** - ***R***) steady-state mRNA transcripts. K-1735 cells isolated from ***brain*** lesions and implanted into the subcutis or grown in culture lose ***MSH*** - ***R*** transcripts and become amelanotic, in contrast to K-1735 cells, B16-BL6 melanoma cells constitutively produce melanin and express high levels of ***MSH*** - ***R*** mRNA regardless of the site of growth. Somatic cell hybrids between K-1735 and B16 cells produced melanin and expressed high levels of ***MSH*** - ***R*** mRNA transcripts, regardless of the site of growth, suggesting the dominance of the B16 phenotype. Treatment with alpha-MSH failed to up-regulate ***MSH*** - ***R*** expression in cultured K-1735 cells or to maintain ***MSH*** - ***R*** expression in K-1735 cells isolated from ***brain*** metastases to be grown in culture. Responsiveness to alpha-MSH as determined by cell proliferation, melanin production, and intracellular accumulation of cyclic AMP directly correlated with ***MSH*** - ***R*** expression. These data demonstrate that a specific organ environment influences the phenotype of metastatic cells by regulation of specific genes that encode for cell surface receptors.

L7 ANSWER 8 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1994:127660 BIOSIS
DN PREV199497140660
TI Plasma concentrations of immunoreactive proopiomelanocortin peptides and cortisol in clinically normal cats.
AU Peterson, Mark E. (1); Kemppainen, Robert J.; Orth, David N.
CS (1) Dep. Med., Anim. Med. Cent., New York, NY 10021 USA
SO American Journal of Veterinary Research, (1994) Vol. 55, No. 2, pp. 295-300. ISSN: 0002-9645.
DT Article
LA English
AB We measured immunoreactive (IR) plasma concentrations of the proopiomelanocortin (POMC)-derived peptides (adrenocorticotropic hormone (ACTH), beta-endorphin/beta-lipotropin (beta-END/beta-LPH), and alpha-melanocyte stimulating hormone ((alpha-MSH)) and of cortisol in 100 clinically normal cats. Median plasma concentration of IR-ACTH was 2.7 pmol/L (range, Itoreq 1.1 to 22 pmol/L), of beta-END/beta-LPH was 28 pmol/L (range, 3.8 to 130 pmol/L), of alpha-MSH was 36 pmol/L (range, It 3.6 to 200 pmol/L), and of cortisol was 35 nmol/L (range, 5 to 140 nmol/l). Plasma concentrations of IR-ACTH, alpha-MSH, and beta-END/beta-LPH were at or below the assay sensitivity in 34, 3, and 0% of the cats, respectively.

We did not detect a correlation between plasma concentrations of IR-ACTH and beta-END/beta-LPH (r = 0 23) or between plasma concentrations of IR-ACTH and alpha- ***MSH*** (***r*** = 0 19) However, there was a significant (P lt 0 001) correlation between plasma concentrations of IR-beta-END/beta-LPH and alpha- ***MSH*** (***r*** = 0 81) There was not a significant correlation between plasma concentration of cortisol and plasma concentration of any of the IR-POMC peptides High plasma concentrations of IR-alpha-MSH and beta-END, POMC peptides secreted predominantly by melanotrophs in other species, indicate that clinically normal cats have an actively secreting pars intermedia Although the beta-END/beta-LPH assay used in this study measures the pars distalis-derived peptide beta-LPH, as well as beta-END itself over 95% of the IR-beta-END/beta-LPH activity in feline plasma containing high concentrations of alpha-MSH, but low concentrations of IR-ACTH, was found to coelute with human beta-END on gel filtration chromatography In contrast to the high plasma concentrations of IR-alpha-MSH and beta-END/beta-LPH, many cats had low to undetectable concentrations of IR-ACTH, a peptide secreted predominantly by pars distalis corticotrophs The pattern of plasma POMC peptide concentrations found in cats is similar to that reported in rats, but is markedly different from that reported in dogs, in which the secretion of pars intermedia POMC peptides is normally low

L7 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1994:450366 CAPLUS
DN 121:50366
TI Cloning and expression of a new member of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** family
AU Barrett, P ; MacDonald, A ; Helliwell, R ; Davidson, G.; Morgan, P
CS Mol Neuroendocrinol Group, Rowett Res Inst , Bucksburn/Aberdeen, AB2 9SB, UK
SO J Mol Endocrinol (1994), 12(2), 203-13
CODEN: JMLEEI, ISSN 0952-5041
DT Journal
LA English
AB A new member of the G protein-coupled receptor superfamily has been isolated from an ovine genomic library with a probe generated by the application of the PCR technique, using cDNA synthesized on a mRNA template isolated from the ovine pars tuberalis This genomic clone encodes a novel receptor of 325 amino acids with seven transmembrane domains These domains share homol with other members of this family, but the best homol is with the recently cloned human MC-1 (50% in the transmembrane domains) and MC-3 (69% in the transmembrane domains) MSH receptors and the human ACTH (42% in the transmembrane domains) receptor When this receptor was expressed in Cos7 cells, it was able to bind a potent analog of .alpha.-MSH, [Nle4,D-Phe7]-.alpha.-MSH (NDP-MSH), with high affinity This binding could be displaced by pro-opiomelanocortin-derived and related peptides, with the order of potency NDP-MSH > .alpha.-MSH = ACTH > .beta.-MSH and with no effect on .gamma.-MSH, .delta.-MSH or .beta.-endorphin The expressed receptor was demonstrated to be functionally coupled to the adenylate cyclase second messenger pathway, with .alpha.-MSH, .beta.-MSH and ACTH stimulating cAMP prodn The amt of the mRNA for this receptor was found to be very low The tissue distribution of this receptor could only be obsd using the reverse transcription-PCR technique and the receptor was found to be present in a no of somatic tissues These data indicate that this is a new and distinct member of the melanocortin receptor family.

L7 ANSWER 10 OF 16 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1994:288152 BIOSIS
DN PREV199497301152
TI Transcriptional induction of the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** alpha- ***MSH*** - ***R*** in K1735 metastatic murine melanoma cells growing in the ***brain*** of syngeneic mice.
AU Radinsky, R. (1); Tsan, R.; Petty, C. M.; Zhang, R.; Price, J. E.; Cone, R.; Fidler, I. J.
CS (1) Dep. Cell Biol., U.T.M.D. Anderson Cancer Cent., Houston, TX 77030 USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1994) Vol. 35, No. 0, pp. 62.
Meeting Info: 85th Annual Meeting of the American Association for Cancer Research San Francisco, California, USA April 10-13, 1994
ISSN: 0197-016X
DT Conference
LA English

L7 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1993:533869 CAPLUS
DN 119:133869
TI Synthetic peptides corresponding to the calmodulin-binding domains of skeletal muscle myosin light chain kinase and human erythrocyte calcium pump interact with and permeabilize liposomes and cell membranes
AU Eshel, Yoav, Shai, Yechiel, Vorherr, Thomas, Carafoli, Ernesto, Salomon, Yoram
CS Dep Horm Res , Weizmann Inst Sci , Rehovot, 76100, Israel
SO Biochemistry (1993), 32(26), 6721-8
CODEN: BICHAW, ISSN: 0006-2960
DT Journal
LA English
AB Synthetic calmodulin-binding (CaM-binding) peptides (CBPs) representing CaM-binding domains of Ca2+/CaM-dependent enzymes have been reported to interfere with the activity of the MSH (MSH) receptor function in melanoma cells (Gerst, J. E., Salomon, Y., 1988) It was postulated that membrane lipids may play an important role in the mode of action of CBPs on cells Therefore the ability of CBPs to interact with membrane bilayers was tested Using artificial phospholipid vesicles, or M2R melanoma cells and cell membranes derived therefrom, as models, it is reported here that synthetic peptides representing the CaM-binding domains of skeletal muscle myosin light chain kinase (M5) and the human erythrocyte calcium pump (C28W), as well as other CBPs, interact with lipid bilayers and cell membranes Significant interactions of CBPs with the lipid bilayer were detected in both model systems M5 and C28W were found to partition into the lipid bilayer of melanoma cell membranes and soybean lecithin vesicles, and surface partition consts obtained (for the liposome model) were in the range 103-104 M-1 In addn , C28W and its N-modified NBD deriv were found to inhibit [125I]iodo-[Nle4,D-Phe7] alpha MSH binding to cultured M2R melanoma cells These and other CBPs were also found to induce the release of cations and calcein from liposomes, suggesting that the interaction of CBPs with the lipid bilayer increases membrane permeability Nonrelevant peptides used as controls were found ineffective Melittin, a bee venom derived CBP, and pardaxin, a shark-repellent ***neurotoxin*** , both membrane-permeating peptides, were in comparison more potent than the enzyme-derived CBPs that were not lytic when applied to cells It is proposed that the tested CBPs act as permeators that partition into the lipid bilayer of the cell membrane, thereby also promoting their interaction with hydrophobic domains of membrane proteins such as the MSH receptor, consequently eliciting the obsd cellular responses

L7 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1993:441147 CAPLUS
DN 119:41147
TI The cloning of a family of genes that encode the melanocortin receptors
AU Mountjoy, Kathleen G., Robbins, Linda S., Mortrud, Marty T., Cone, Roger D.
CS Vollum Inst. Adv. Biomed. Res., Oregon Health Sci. Univ., Portland, OR, 97201, USA
SO Science (Washington, D. C., 1883-) (1992), 257(5074), 1248-51
CODEN: SCIEAS, ISSN: 0036-8075
DT Journal
LA English
AB MSH and adrenocorticotropic hormone (ACTH) regulate pigmentation and adrenal cortical function, resp. These peptides also have a variety of biol. activities in other areas, including the ***brain*** , the pituitary, and the immune system. A complete understanding of the biol. activities of these hormones requires the isolation and characterization of their corresponding receptors. The murine and human MSH receptors (MSH-Rs) and a human ACTH receptor (ACTH-R) were cloned. These receptors
define a subfamily of receptors coupled to guanine nucleotide-binding proteins that may include the cannabinoid receptor.

L7 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1974:116509 CAPLUS
DN 80:116509
TI Estrogen influence on the hypothalamic enzymes involved in the formation of melanocyte-stimulating hormone release-inhibiting factor (***MSH*** - ***R*** -IF)
AU Celis, Maria E., Taleisnik, S
CS Inst. Invest. Med. Mercedes y Martin Ferreyra, Cordoba, Argent.
SO Proc. Soc. Exp. Biol. Med. (1974), 145(1), 142-4
CODEN: PSEBAA
DT Journal
LA English
AB The activity of an enzyme which yielded ***MSH*** - ***R*** -IF (MSH release inhibiting factor) [9061-59-0] upon incubation of rat hypothalamic exts. with oxytocin was estrogen sensitive. The enzyme was absent in ovariectomized rats and increased 10-fold after the s.c. administration of 10 .mu.g estradiol benzoate (I) [50-50-0]. Cycloheximide and actinomycin D inhibited the effect of estrogen, suggesting that estrogen promotes the formation of new enzyme.

L7 ANSWER 14 OF 16 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V. DUPLICATE 5
AN 74082573 EMBASE
DN 1974082573
TI Interaction between paraventricular nucleus extracts and median eminence extracts on the formation of melanocyte stimulating hormone release inhibiting factor
AU Celis M.E.; Taleisnik S.
CS Inst. Invest. Med. Mercedes Y Martin Ferreyra, Cordoba, Argentina
SO Life Sciences, (1973) 13/5 (493-499)
CODEN: LIFSAK
DT Journal
FS 003 Endocrinology
008 Neurology and Neurosurgery
030 Pharmacology
LA English
AB Median eminence extracts (ME(incubated with oxytocin produced the formation of a melanocyte stimulating hormone release inhibiting factor (***MSH*** ***R*** IF) When extracts of paraventricular nucleus (PVN) were added to that system and incubated together no formation of ***MSH*** ***R*** IF was detected The effect of PVN extracts is due to the inactivation of ***MSH*** ***R*** IF, and is proportional to the amount of extract used PVN extracts were effective in preventing

the formation of ***MSH*** ***R*** IF when 150 mU oxytocin were used as substrate but not when 1000 mU were used, suggesting that the mechanism by which the blocking effect is exerted is similar to that of two enzymes competing for the same substrate It is postulated that ***MSH*** ***R*** IF formation depends on two hypothalamic agents of antagonistic influence acting on oxytocin

L7 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1972:456983 CAPLUS
DN 77:56983
TI MSH-release-inhibiting factor: Inactivation by proteolytic enzymes
AU Marks, Neville; Walter, Roderich
CS New York State Res. Inst. Neurochem. Drug Addict., New York, N. Y., USA
SO Proc. Soc. Exp. Biol. Med. (1972), 140(2), 673-6
CODEN: PSEBAA
DT Journal
LA English
AB Leucine aminopeptidase(E.C. 3.4.1.1), aminopeptidase M, thermolysin, and ***brain*** arylamidase degraded melanocyte-stimulating hormone-release-inhibiting factor (***MSH*** - ***R*** -IF), but collagenase(E.C. 3.4.4.19), carboxypeptidase A(E.C. 3.4.2.1), carboxypeptidase B(E.C. 3.4.2.2), and chymotrypsin A(E.C. 3.4.4.5), did not. Crude rat tissue exts. were also tested for enzymic activity. ***Brain*** extracts were highly active in degrading ***MSH*** - ***R*** -IF, whereas exts. of the median eminence, a storage site for the factor, were significantly less active

L7 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1969:10246 CAPLUS
DN 70:10246
TI Influence of reserpine on the content of pituitary melanocyte-stimulating hormone and on hypothalamic factors which affect its release
AU Tomatis, Maria E.; Taleisnik, Samuel
CS Inst. Invest. Med. Mercedes y Martin Ferreyra, Cordoba, Argent.
SO J. Endocrinol. (1968), 42(4), 505-12
CODEN: JOENAK
DT Journal
LA English
AB The MSH content of toad pituitary glands was decreased by treatment with reserpine (0.2 mg./kg./day s.c.). The hormone content remained low for 1 week but regained normal levels after 2 weeks. All injected animals showed intense darkening of the skin. In rats, a drop in pituitary MSH content also occurred after reserpine treatment but normal values were found after 7-14 days of treatment. MSH-releasing factor found in stalk-median eminence tissue of normal male rats was not present in the reserpine-injected animals, but after 7 days of treatment an increase in MSH-release-inhibiting factor (***MSH*** - ***R*** -IF) was demonstrated. ***MSH*** - ***R*** -IF was also increased in female castrated rats after 2 days of treatment with reserpine. It was concluded that reserpine permits the secretion of pituitary MSH by blocking the release of ***MSH*** - ***R*** -IF, which accumulates in the hypothalamic ***neurons***

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NEWS 13 Nov 30 Files VETU and VETB to have open access
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NEWS 18 Dec 17 New fields for DPCI
NEWS 19 Dec 19 CAS Roles modified
NEWS 20 Dec 19 1907-1946 data and page images added to CA and CAplus
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NEWS 23 Jan 29 FSTA has been reloaded and moves to weekly updates
NEWS 24 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 25 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 26 Mar 08 Gene Names now available in BIOSIS

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L3 ANSWER 1 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2001:270133 BIOSIS
DN PREV200100270133
T Genetic factors determining cutaneous basal cell carcinoma phenotype
AU Ramachandran, Sudarshan; Fryer, Anthony A.; Strange, Richard C. (1)
CS (1) Centre for Cell and Molecular Medicine, School of Postgraduate Medicine, Keele University, North Staffordshire Hospital, Stoke-on-Trent, Staffordshire paa00@keele.ac.uk UK
SO Medical and Pediatric Oncology, (May, 2001) Vol. 36, No. 5, pp. 559-563. print.
ISSN: 0098-1532
DT Article
LA English

SL English
AB Background Basal cell carcinoma (BCC) patients demonstrate considerable phenotypic diversity The basis of this heterogeneity is poorly understood We have shown that presentational phenotypes are associated with BCC numbers Thus, patients with a cluster of new BCC at any presentation comprise a subgroup termed MPP, that is at increased risk of developing numerous lesions Patients with more than one cluster (multiple cluster MPP) are at particular risk Procedure We determined in a cohort of BCC cases, whether (i) ***tumor*** accrual was altered after clustering, and (ii) multiple cluster MPP is associated with characteristics linked with sensitivity to UV or, GSTT1, GSTM1, GSTM3, GSTP1, MC1R, CYP2D6, TNF-alpha, and VDR genotypes previously associated
with BCC presentational phenotypes Results (i) After clustering BCC accrual increased, and (ii) exposure to UV in single and multiple cluster MPP cases were similar In multiple cluster cases, mean age at first presentation with a single ***tumor*** occurred earlier and, the frequencies of CYP2D6 EM (94 4%) and GSTT1 null (41 2%) were significantly greater (P = 0 028 and P = 0 004) than in single cluster cases (67 1 and 14 3%) The odds ratios for these associations with the multiple cluster MPP were large, 15 5 and 7 39, respectively Conclusions The finding of clusters of new, primary BCC is a critical event that is followed by markedly increased accrual of further ***tumors*** Clustering occurs at a relatively late age and may be associated with a failure in immune surveillance We propose the MPP is not the consequence of excessive UV exposure but reflects the presence of a distinct BCC subgroup defined by a combination of risk genes

L3 ANSWER 2 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 2001 352473 BIOSIS
DN PREV200100352473
TI Radiometallated receptor-avid peptide conjugates for specific in vivo targeting of ***cancer*** cells
AU Hoffman, T J ; Quinn, T P ; Volkert, W A (1)
CS (1) Department of Radiology, University of Missouri and Research Service, Columbia, MO, 65211 VolkertW@health.missouri edu USA
SO Nuclear Medicine and Biology, (July, 2001) Vol 28, No 5, pp 527-539 print.
ISSN 0969-8051
DT General Review
LA English
SL English
AB New receptor-avid radiotracers are being developed for site-specific in vivo targeting of a myriad of receptors expressed on ***cancer*** cells This review exemplifies strategies being used to design radiometallated peptide conjugates that maximize uptake in ***tumors*** and optimize their in vivo pharmacokinetic properties Efforts to produce synthetic peptide analogues that target the following three receptor systems are highlighted Gastrin releasing peptide (GRP), alpha-melanocyte stimulating hormone (alpha-MSH), and guanylate cyclase-C (GC-C) receptors

L3 ANSWER 3 OF 25 CAPLUS COPYRIGHT 2002 ACS
AN 1999:589640 CAPLUS
DN 131 309750
TI Receptor-mediated modulation of murine mast cell function by .alpha.-melanocyte stimulating hormone
AU Adachi, Shiro; Nakano, Teruaki, Vliagoftis, Harrisios; Metcalfe, Dean D
CS Laboratory of Allergic Disease, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD, 20892, USA
SO J Immunol (1999), 163(6), 3363-3368
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English
AB The proopiomelanocortin (POMC)-derived neuropeptide alpha -MSH is known to modulate some aspects of inflammation through direct effects on T cells, B cells, and monocytes To det whether alpha.-MSH might similarly influence mast cell responsiveness, mast cells were examd to see if they expressed the receptor for alpha -MSH, melanocortin-1 (MC-1), and whether alpha -MSH altered mast cell function The authors thus first identified MC-1 on bone marrow cultured murine mast cells (BMCMC) and a murine mast cell line (MCP-5) employing flow cytometry and through detection of specific binding Subsequent treatment of mast cells with .alpha.-MSH increased the cAMP concn in a characteristic biphasic pattern, demonstrating that alpha -MSH could affect intracellular processes The authors next examd the effect of .alpha.-MSH on mediator release and cytokine expression IgE/DNP-human serum albumin-stimulated histamine release from mast cells was inhibited by apprx 60% in the presence of alpha -MSH Although activation of BMCMC induced the expression of mRNAs for the inflammatory cytokines IL-1 beta , IL-4, IL-6, TNF- alpha , and the chemokine lymphotactin, mRNAs for IL-1 beta , TNF- alpha , and lymphotactin were down-modulated in the presence of .alpha -MSH Finally, IL-3-dependent proliferative activity of BMCMC was slightly but significantly augmented by alpha -MSH Thus, alpha -MSH may exert an inhibitory effect on the mast cell-dependent component of a specific inflammatory response
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L3 ANSWER 4 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1999 343142 BIOSIS
DN PREV199900343142
TI alpha-MSH and its receptors in regulation of ***tumor*** necrosis factor-alpha production by human monocyte/macrophages
AU Taherzadeh, S ; Sharma, S ; Chhajlani, V ; Gantz, I ; Rajora, N ; Demitri, M T ; Kelly, L ; Zhao, H ; Ichiyama, T ; Catania, A ; Lipton, J M (1)
CS (1) Univ of Texas Southwestern Medical Center, 5323 Harry Hines Blvd , Dallas, TX, 75235-9040 USA
SO American Journal of Physiology, (May, 1999) Vol 276, No 5 PART 2, pp R1289-R1294
ISSN 0002-9513
DT Article
LA English
SL English
AB The hypothesis that macrophages contain an autocrine circuit based on melanocortin (ACTH and alpha-melanocyte-stimulating hormone (alpha-MSH)) peptides has major implications for neuroimmunomodulation research and inflammation therapy To test this hypothesis, cells of the THP-1 human monocyte/macrophage line were stimulated with lipopolysaccharide (LPS) in the presence and absence of alpha-MSH The inflammatory cytokine ***tumor*** necrosis factor (TNF)-alpha was inhibited in relation to alpha-MSH concentration Similar inhibitory effects on TNF-alpha were observed with ACTH peptides that contain the alpha-MSH amino acid sequence and act on melanocortin receptors Nuclease protection assays indicated that expression of the human melanocortin-1 receptor subtype (hMC-1R) occurs in THP-1 cells, Southern blots of RT-PCR product revealed that additional subtypes, hMC-3R and hMC-5R, also occur Incubation of resting macrophages with antibody to hMC-1R increased TNF-alpha concentration; the antibody also markedly reduced the inhibitory influence of alpha-MSH on TNF-alpha in macrophages treated with LPS These results in cells known to produce alpha-MSH at rest and to increase secretion of the peptide when challenged are consistent with an endogenous regulatory circuit based on melanocortin peptides and their receptors Targeting of this neuroimmunomodulatory circuit in inflammatory diseases in which myelomonocytic cells are prominent should be beneficial

L3 ANSWER 5 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE
1
AN 1999 366170 BIOSIS
DN PREV199900366170
TI Association of NAD(P)H quinone oxidoreductase (NQO1) null with numbers of basal cell carcinomas: Use of a multivariate model to rank the relative importance of this polymorphism and those at other relevant loci
AU Clairmont, Annette; Sies, Helmut; Ramachandran, Sudarshan; Lear, John T ; Smith, Andrew G ; Bowers, Bill; Jones, Peter W ; Fryer, Anthony A ; Strange, Richard C (1)
CS (1) Clinical Biochemistry Research Laboratory, School of Postgraduate Medicine, Centre for Cell and Molecular Medicine, Keele University, North Staffordshire Hospital, Hartshill, Stoke-on-Trent, Staffordshire, ST4 7QB UK
SO Carcinogenesis (Oxford), (July, 1999) Vol 20, No 7, pp 1235-1240
ISSN 0143-3334
DT Article
LA English
SL English
AB Glutathione S-transferase GSTM1 B and GSTT1 null, and cytochrome P450 CYP2D6 EM have been associated with cutaneous basal cell carcinoma (BCC) numbers, although their quantitative effects show that predisposition to many BCC is determined by an unknown number of further loci We speculate that other loci that determine response to oxidative stress, such as NAD(H):quinone oxidoreductase (NQO1) are candidates Accordingly, we assessed the association between NQO1 null and BCC numbers primarily to rank NQO1 null in a model that included genotypes already associated with BCC numbers We found that only 14 out of 457 cases (3 1%) were NQO1 null This frequency did not increase in cases with characteristics linked with BCC numbers including gender, skin type, a truncal lesion or more than one new BCC at any presentation (MPP) However, the mean number of BCC in NQO1*0 homozygotes was greater than in wild-type allele homozygotes and heterozygotes, although the difference was not quite significant (P = 0 06) These data reflect the link between NQO1 null and BCC numbers in the 42 MPP cases rather than the whole case group We identified an interaction between NQO1 null and GSTT1 null that was associated with more BCC (P = 0 04), although only four cases had this combination The relative influence of NQO1 null was studied in a multivariate model that included: (i) 241 patients in whom GSTM1 B, GSTT1 null and CYP2D6 EM genotype data were available, and (ii) 101 patients in whom these genotypes, as well as data on GSTM3, CYP1A1 and ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1R) genotypes were available NQO1 null (P = 0 001) and MC1R asp294/asp294 (P = 0 03) were linked with BCC numbers, and the association with CYP2D6 EM approached significance (P = 0 08) In a stepwise regression model only these genotypes were significantly associated with BCC numbers with NQO1 null being the most powerful predictor

L3 ANSWER 6 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE
2
AN 1999 494936 BIOSIS
DN PREV199900494936
TI Cytochrome P450 CYP2D6 genotypes Association with hair colour Breslow

thickness and ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** alleles in patients with malignant melanoma

AU Strange, Richard C. (1); Ellison, Tracy; Ichii-Jones, Fumiyo; Bath, Joanna; Hoban, Paul; Lear, John T.; Smith, Andrew G.; Hutchinson, Peter E.; Osborne, Joy; Bowers, Bill; Jones, Peter W.; Fryer, Anthony A.

CS (1) School of Postgraduate Medicine, Keele University, North Staffordshire Hospital, Stoke-on-Trent, Staffordshire, ST4 7PA UK

SO Pharmacogenetics, (June, 1999) Vol. 9, No. 3, pp. 269-276. ISSN: 0960-314X.

DT Article

LA English

SL English

AB We previously identified associations between polymorphism in the cytochrome P450 CYP2D6 gene and outcome in several ***cancers***. We have now examined the hypothesis that homozygosity for the mutant alleles, CYP2D6*4 and CYP2D6*3, is associated with susceptibility and outcome in malignant melanoma. Outcome was assessed by Breslow thickness. We first confirmed previous reports that these mutant alleles are associated with increased susceptibility to malignant melanoma. For example, the frequency of homozygosity for CYP2D6*4 was significantly greater (P = 0.006, chi-squared 1 d.f. = 7.4, odds ratio 2.2, 95% confidence interval 1.2, 3.9) in cases (9.1%) than in control individuals (4.3%). The frequency of homozygosity for the mutant alleles was next examined in the malignant melanoma cases grouped on the basis of characteristics associated with malignant melanoma risk. Homozygosity was significantly more common (P = 0.038) in cases with red/blonde hair than in those with brown/black hair. We found no associations between the CYP2D6 genotype and sex, skin type or eye colour. The possible association of CYP2D6 with outcome was assessed by comparing genotype frequencies in patients with tumours of Breslow thickness < 1.5 mm with those whose tumours were gtoreq 1.5 mm. In patients with red/blonde, but not brown or black hair, homozygosity for CYP2D6*4 was significantly associated with thicker lesions in a multivariate model (P = 0.036). We further examined the association of CYP2D6*4 homozygosity with red/blonde hair by classifying patients on the basis of homo- or heterozygosity for wild-type or val92met, asp294his or asp84glu ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1R) alleles. None of the nine patients with brown/black hair with the asp294his allele were homozygotes for CYP2D6*4. By contrast, in the patients with red/blonde hair, three of five cases with asp294his were homozygotes for the mutant CYP2D6 allele. The difference in the frequency of CYP2D6*4 homozygotes in the red/blonde cases with wild-type MC1R alleles compared with those with asp294his was significant (exact P = 0.029). No associations between val92his or asp84glu and CYP2D6 alleles were identified.

L3 ANSWER 7 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 3

AN 1999:293085 BIOSIS

DN PREV199900293085

TI The ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** polymorphism: Association of the V92M and A294H alleles with basal cell carcinoma

AU Jones, Fumiyo Ichii; Ramachandran, Sudarshan; Lear, John; Smith, Andrew; Bowers, Bill; Ollier, William E.R.; Jones, Peter; Fryer, Anthony A.; Strange, Richard C. (1)

CS (1) Clinical Biochemistry Research Group, School of Postgraduate Medicine, North Staffordshire Hospital, Keele University, Stoke-on-Trent, Staffordshire UK

SO Clinica Chimica Acta, (April, 1999) Vol. 282, No. 1-2, pp. 125-134. ISSN: 0009-8981.

DT Article

LA English

SL English

AB Allelic variants in the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1R) gene are susceptibility/outcome candidates for cutaneous basal cell carcinoma (BCC). We identified the val92met (V92M) and asp294his (A294H) alleles in 311 cases and 190 controls. The cases included four homo- and 53 heterozygotes for V92M and 12 heterozygotes for A294H and two compound heterozygotes (V92M/A294H). Allele frequencies were similar in controls. In the cases, we found no association between the alleles and skin type though A294H was more common in those with red hair (4/19) than with other hair colours (6/163) (P = 0.012). V92M was not associated with BCC numbers. Cases with A294H had fewer BCC in comparison with those without the allele though the difference was not significant. After inclusion of red hair in the model, A294H was significantly associated with fewer tumours. While MC1R alleles are attractive candidates for BCC, the variants studied did not influence susceptibility. The association with outcome was relatively weak. The large number of MC1R alleles and their low frequency, make assessment of the importance of this gene in the pathogenesis of skin ***cancers*** difficult.

L3 ANSWER 8 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

AN 1999:6018 BIOSIS

DN PREV199900006018

TI Design and characterization of alpha-melanotropin peptide analogs cyclized through rhenium and technetium metal coordination.

AU Giblin, Michael F.; Wang, Nannan; Hoffman, Timothy J.; Jurisson, Silvia S.; Quinn, Thomas P. (1)

CS (1) Dep. Biochemistry, 117 Schweitzer Hall, Univ. Missouri, Columbia, MO 65211 USA

SO Proceedings of the National Academy of Sciences of the United States of America, (Oct. 27, 1998) Vol. 95, No. 22, pp. 12814-12818. ISSN: 0027-8424.

DT Article

LA English

AB alpha-Melanocyte stimulating hormone (alphaMSH) analogs cyclized through site-specific rhenium (Re) and technetium (Tc) metal coordination were structurally characterized and analyzed for their abilities to bind alpha-MSH receptors present on melanoma cells and in ***tumor*** -bearing mice. Results from receptor-binding assays conducted with B16-F1 murine melanoma cells indicated that receptor-binding affinity was reduced to approximately 1% of its original levels after Re incorporation into the cyclic CyS4,10, D-Phe7-alpha-MSH4-13 analog. Structural analysis of the Re-peptide complex showed that the disulfide bond of the original peptide was replaced by thiolate-metal-thiolate cyclization. A comparison of the metal-bound and metal-free structures indicated that metal complexation dramatically altered the structure of the receptor-binding core sequence. Redesign of the metal binding site resulted in a second-generation Re-peptide complex (ReCCMSH) that displayed a receptor-binding affinity of 2.9 nM, 25-fold higher than the initial Re-alpha-MSH analog. Characterization of the second-generation Re-peptide complex indicated that the peptide was still cyclized through Re coordination, but the structure of the receptor-binding sequence was no longer constrained. The corresponding 99mTc- and 188ReCCMSH complexes were synthesized and shown to be stable in phosphate-buffered saline and to challenges from diethylenetriaminepentaacetic acid (DTPA) and free cysteine. In vivo, the 99mTcCCMSH complex exhibited significant ***tumor*** uptake and retention and was effective in imaging melanoma in a murine- ***tumor*** model system. Cyclization of alpha-MSH analogs via 99mTc and 188Re yields chemically stable and biologically active molecules with potential melanoma-imaging and therapeutic properties.

L3 ANSWER 9 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC

AN 1998:376047 BIOSIS

DN PREV199800376047

TI Malignant melanoma skin type and polymorphism in the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** gene.

AU Lear, J. (1); Jones, F.; Smith, A.; Hutchinson, P.; Osbourne, J.; Ollier, W.; Fryer, A.; Strange, R. C.

CS (1) Dep. Dermatol., Bristol Royal Infirmary, Bristol UK

SO British Journal of Dermatology, (April, 1998) Vol. 138, No. 4, pp. 746. Meeting Info.: Annual Meeting of the British Society for Investigative Dermatology and British Photodermatology Group Liverpool, England, UK April 22-24, 1998 British Photodermatology Group. ISSN: 0007-0963.

DT Conference

LA English

L3 ANSWER 10 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 4

AN 1998:405474 BIOSIS

DN PREV199800405474

TI Susceptibility to melanoma: Influence of skin type and polymorphism in the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** gene.

AU Ichii-Jones, Fumiyo; Lear, John T.; Heagerty, Adrian H. M.; Smith, Andrew G.; Hutchinson, Peter E.; Osborne, Joy; Bowers, Bill; Jones, Peter W.; Davies, Eric; Ollier, William E. R.; Thomson, Wendy; Yengi, Lillian; Bath, Joanna; Fryer, Anthony A.; Strange, Richard C. (1)

CS (1) Sch. Postgraduate Med., Univ. Keele, North Staffordshire Hosp., Stoke-on-Trent, Staffordshire ST4 7PA UK

SO Journal of Investigative Dermatology, (Aug., 1998) Vol. 111, No. 2, pp. 218-221. ISSN: 0022-202X.

DT Article

LA English

AB Allelic variation at the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** (MC1R) gene has been linked with sun-sensitive skin types, suggesting it is a susceptibility candidate for melanoma. We determined the frequency of the val92met, asp294his, and asp84glu MC1R alleles in 190 Caucasian controls and 306 melanoma cases and studied their association with skin type and hair color. The percentage of controls with at least one val92met, asp294his, or asp84glu allele was 17.3%, 6.8%, and 3.5%, respectively. Individually, frequencies of the val92met, asp294his, or asp84glu alleles in the controls with skin types 3 and 4 were similar to those with skin types 1 and 2. Trend analysis, however, did identify an association (exact p = 0.048, two-sided test) between skin type and MC1R variants in the group comprising all controls with any one or more of these alleles. There was no association between MC1R alleles and hair color. Allele frequencies were not different in melanoma cases and controls. There were no associations between skin types and the proportion of cases with the asp294his or asp84glu alleles, though the association between skin type and the val92met allele approached significance (exact p = 0.09, two-sided test). Unexpectedly, in the group comprising all cases with one or more variant alleles, the proportion of subjects with variant alleles increased with skin types associated with tanning rather than burning, although trend analysis showed that this association did not quite reach statistical significance (exact p = 0.08, two-sided test). Asp84glu (but not val92met or asp294his) variant alleles

were more common in subjects with blonde hair, although the relationship between the asp84glu allele and hair color did not achieve statistical significance (chi32 = 6 16, exact p = 0 10) We interpret the data presented as indicating that polymorphism at MC1R does not appear a major determinant of skin type, at least in terms of these allelic variants Furthermore, considered alone, these alleles are not susceptibility candidates for malignant melanoma

L3 ANSWER 11 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE
5
AN 1998 434529 BIOSIS
DN PREV199800434529
TI Modulation of ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** expression on normal human melanocytes Evidence for a regulatory role of ultraviolet B interleukin-1alpha, interleukin-1beta, endothelin-1 and tumour necrosis factor-alpha
AU Funasaka, Y (1), Chakraborty, A K, Hayashi, Y, Komoto, M, Ohashi, A Nagahama, M, Inoue, Y, Pawelek, J, Ichihashi, M
CS (1) Dep Dermatol, Kobe Univ Sch Med, 5-1 Kusunoki-cho 7-chome, Chu-ku, Kobe 650 Japan
SO British Journal of Dermatology, (Aug., 1998) Vol. 139, No. 2, pp. 216-224 ISSN: 0007-0963
DT Article
LA English
AB Melanocyte-stimulating hormone (MSH) receptor binding activity and melanocortin-1 receptor (MC1-R) gene expression on normal human melanocytes have been studied as responses to the effects of ultraviolet B (UVB), interleukin-1 (IL-1), endothelin-1 (ET-1) and tumour necrosis factor-alpha (TNF-alpha), which are known as UV sensitive regulators of melanocytic function MSH receptor (***MSH*** - ***R***) binding activity was upregulated by UVB, IL-1alpha, -1beta and ET-1, but was downregulated by TNF-alpha. Northern blot analysis showed that MC1-R mRNA expression was induced 24h after UVB irradiation in a dosedependent manner, and that 24-h treatment with ET-1 also induced an expression of MC1-R mRNA, whereas TNF-alpha downregulated the expression. In addition, IL-1alpha and -1beta have a small but real inductive effect on MC1-R mRNA expression. Taken together, our results suggest a model in which higher MC1-R MRNA expression is accompanied by upregulation of ***MSH*** - ***R*** binding activity, and enhanced by UVB or cytokines sensitive to UVB. Such a regulatory system would enable normal human melanocytes to respond to MSH more efficiently and induce an increase of melanization of the skin through the MSH/ ***MSH*** - ***R*** system after UVB radiation.

L3 ANSWER 12 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE
6
AN 1999.88552 BIOSIS
DN PREV199900088552
TI Human pigmentation genes and their response to solar UV radiation.
AU Sturm, Richard A. (1)
CS (1) Cent. Mol. Cell Biol., Univ. Queensl., Brisbane, Qld. 4072 Australia
SO Mutation Research, (Nov. 9, 1998) Vol. 422, No. 1, pp. 69-76 ISSN: 0027-5107.
DT General Review
LA English
AB Identification and characterisation of the genes involved in melanin pigment formation, together with the study of how their action is influenced by exposure to UV radiation, is providing a molecular understanding of the process of skin photoprotection through tanning. The mechanisms underlying this change in epidermal melanin involve both a transcriptional response of the pigmentation genes and post-translational control of the melanin biosynthetic pathway. UV rays are known to interact with numerous molecules within cells, and among these the photochemical reactions involving lipids and DNA are implicated in modulating melanogenesis. The combination of DNA damage, the formation of diacylglycerol, and the action of the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** are all likely to be involved in UV-induced tanning.

L3 ANSWER 13 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997.489360 BIOSIS
DN PREV199799788563
TI Synthetic peptides derived from the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** MC1R can stimulate HLA-A2-restricted cytotoxic T lymphocytes that recognize naturally processed peptides on human melanoma cells
AU Salazar-Onfray, Flavio, Nakazawa Tsutomu, Chhajlani, Vijay, Petersson, Max, Karre, Klas, Masucci, Giuseppe, Celis, Esteban, Sette, Alessandro, Southwood, Scott, Appella, Ettore, Kiessling, Rolf (1)
CS (1) Microbiol Tumor Biol Cent, Karolinska Inst., S-171 77 Stockholm Sweden
SO Cancer Research, (1997) Vol. 57, No. 19, pp. 4348-4355. ISSN: 0008-5472
DT Article
LA English
AB Human melanoma-specific HLA-A2 restricted CTLs have recently been shown to
recognize antigens expressed by melanoma lines and normal melanocytes, including Melan-A/Mart-1, gp100, gp75, and tyrosinase. Herein, we define HLA-A2-restricted CTL epitopes from a recently cloned melanocortin 1 receptor (MCIR), which belongs to a new subfamily of the G-protein-coupled receptors expressed on melanomas and melanocytes. Thirty-one MCIR-derived peptides were selected on the basis of HLA-A2-specific motifs and tested for their HLA-A2 binding capacity. Of a group of 12 high or intermediate HLA-A2 binding peptides, three nonamers, MCIR244 (TILLGIFFL), MC1R283 (FLALIICNA), and MCIR291 (AIIDPLIYA), were found to induce peptide-specific CTLs from peripheral blood mononuclear cells of healthy HLA-A2+ donors after repeated in vitro stimulation with peptide-pulsed antigen-presenting cells. The CTLs raised against these three HLA-A2+-restricted peptides could recognize naturally processed peptides from HLA-A2+ melanomas and from Cos7 cells cotransfected with MCIR and HLA-A2. CTLs induced by the MCIR291 peptide (but not induced or induced only to a very low extent by the other two MCR1 peptide epitopes) showed cross-reactions with two other members of the melanocortin receptor family, which are more broadly expressed on other tissues. Taken together, our findings have implications in relation both to autoimmunity and immunotherapy of malignant melanomas

L3 ANSWER 14 OF 25 EMBASE COPYRIGHT 2002 ELSEVIER SCI B.V.
AN 97017690 EMBASE
DN 1997017690
TI Identification of common polymorphisms in the coding sequence of the human MSH receptor (MCIR) with possible biological effects
AU Koppula S.V., Robbins L.S., Lu D., Baack E., White C.R. Jr., Swanson N.A., Cone R.D.
CS R.D. Cone, VIABR, Portland, OR 97201, United States
SO Human Mutation, (1997) 9/1 (30-36).
Refs: 18
ISSN: 1059-7794 CODEN: HUMUE3
CY United States
DT Journal; Article
FS 013 Dermatology and Venereology
016 Cancer
022 Human Genetics
LA English
SL English
AB The extension locus has been identified in many mammalian species as a gene that determines the relative amounts of eumelanin and phaeomelanin pigments in hair and skin. In at least three species, this locus has been demonstrated to encode the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1-R), and functionally variant alleles have been demonstrated to cause a broad range of pigmentation phenotypes. To test for MC1-R allelic variation in man, genomic DNA was extracted from skin samples collected from patients with different skin types (I-VI), and eye and hair color. A PCR-based approach was used to amplify the full-length coding sequence of the MC1-R and the resulting products were sequenced. Two polymorphic alleles were identified with single point mutations in the coding sequence: a valine-to-methionine substitution at position 92 (V92M), and an aspartic acid-to-glutamic acid substitution at position 84 (D84E). RFLP analysis demonstrated the presence of the V92M allele in 4 out of 60 (6.6%) of individuals examined, predominantly those with blue eyes and blond hair. This polymorphism was found in both heterozygous and homozygous states in individuals with type I skin. The D84E allele was found in one individual with skin type I; this person also has the V92 M allele and thus is a compound heterozygote.

L3 ANSWER 15 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1998 106967 BIOSIS
DN PREV199800106967
TI Vaccination against ***cancer*** ; a daydream of a scientist or a future reality in the clinic.
AU Kiessling, Rolf
SO Immunology. (Dec., 1997) Vol. 92, No. SUPPL. 1, pp. 12
Meeting Info.: 5th Annual Congress of the British Society for Immunology Brighton, England, UK December 2-5, 1997 British Society for Immunology ISSN: 0019-2805.
DT Conference
LA English

L3 ANSWER 16 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997 376308 BIOSIS
DN PREV199799675511
TI Variants of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** gene modify melanoma risk in familial atypical multiple mole-melanoma (FAMMM) syndrome families.
AU Gruis, N. A. (1), Van Der Velden, P. A., Sandkuijl, L. A., Bergman, W., Frants, R. R.
CS (1) Dep. Dermatol., Leiden University Hosp., Leiden Netherlands
SO Melanoma Research, (1997) Vol. 7, No. SUPPL. 1, pp. S9
Meeting Info.: 4th World Conference on Melanoma Sydney, Australia June 10-14, 1997
ISSN: 0960-8931
DT Conference, Abstract
LA English

L3 ANSWER 17 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC
AN 1996 555330 BIOSIS
DN PREV199699277686
TI ***Melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (MC1R) variants modify melanoma risk in Dutch FAMMM

families
AU Frants, R R (1), Van Der Velden, P A (1), Bergman, W , Gruis, N A (1)
CS (1) MGC-Dep Human Genetics, Leiden Univ , Leiden Netherlands
SO American Journal of Human Genetics, (1996) Vol 59, No 4 SUPPL , pp A67 Meeting Info 46th Annual Meeting of the American Society of Human Genetics San Francisco, California, USA October 29-November 2 1996 ISSN 0002-9297
DT Conference
LA English

L3 ANSWER 18 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 7
AN 1995 510421 BIOSIS
DN PREV199598515471
TI Agouti antagonism of melanocortin binding and action in the B-16F-10 murine melanoma cell line
AU Blanchard, Steven G (1), Harris, Cole O , Ittoop, Olivia R R , Nichols, James S , Parks, Derek J , Truesdale, Anne T , Wilkison, William O
CS (1) Dep Biochem Mol Sci , Glaxo Res Inst , Glaxo Inc , 5 Moore Drive, Research Triangle Park, NC 27709 USA
SO Biochemistry, (1995) Vol 34, No 33, pp 10406-10411 ISSN 0006-2960
DT Article
LA English
AB Several dominant mutations at the murine agouti locus result in the expression of a number of phenotypic changes, including a predominantly yellow coat color, obesity, and hyperinsulinemia The mutants exhibit ectopic overexpression of normal agouti protein, suggesting that agouti regulates coat coloration by direct antagonism of the alpha-***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** We have tested this hypothesis by examining agouti inhibition of both melanocortin-stimulated cyclic adenosine monophosphate production and the binding of a radioactive melanocortin analog in the murine B-16F-10 melanoma cell line. Inhibition of melanocortin-induced cyclic nucleotide accumulation did not require preincubation of the cells with agouti and was independent of the agonist used. Furthermore, inhibition of both agonist binding to and activation of melanocortin receptor could be described by a simple competitive model with similar inhibition constants of 1 9 and 0 9 nM, respectively. The mutually exclusive binding of agouti and melanocortin was verified by cross-linking experiments using a radiolabeled alpha-melanocyte-stimulating hormone analog. Competitive inhibiton of alpha-melanocyte-stimulating hormone binding can account for the effects of agouti on coat coloration and suggests the possibility that the other phenotypic changes observed on agouti overexpression may be due to direct action of agouti at a novel melanocortin receptor(s)

L3 ANSWER 19 OF 25 EMBASE COPYRIGHT 2002 ELSEVIER SCI B.V.
AN 96012299 EMBASE
DN 1996012299
TI Modulation of ***tumor*** cell gene expression and phenotype by the organ-specific metastatic environment.
AU Radinsky R.
CS University of Texas, MD Anderson Cancer Center, 1515 Holcombe Boulevard,Houston, TX 77030, United States
SO Cancer and Metastasis Reviews, (1995) 14/4 (323-328) ISSN: 0167-7659 CODEN: CMRED4
CY United States
DT Journal; General Review
FS 005 General Pathology and Pathological Anatomy
016 Cancer
022 Human Genetics
037 Drug Literature Index
LA English
SL English
AB The mechanistic basis of a metastatic cell's ability to proliferate in the parenchyma of certain organs and develop organ-specific metastases is under intense investigation. Signals from paracrine or autocrine pathways, alone or in combination, may regulate ***tumor*** cell proliferation with the eventual outcome dependent on the net balance of stimulatory and inhibitory factors. This article summarizes recent reports from our laboratory and others demonstrating that the organ microenvironment can profoundly influence the pattern of gene expression and the biological phenotype of metastatic ***tumor*** cells, including induction of ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** and production of melanin, regulation of terminal differentiation and apoptosis, resistance to chemotherapy, and regulation of growth at the organ-specific metastatic site. These recent data from both murine and human ***tumor*** models support the concept that the microenvironment of different organs can influence the pattern of gene expression and hence the phenotype of ***tumor*** cells at different steps of the metastatic process. These findings have obvious implications for the therapy of ***neoplasms*** in general and metastases in particular.

L3 ANSWER 20 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC DUPLICATE 8
AN 1995 110714 BIOSIS
DN PREV199598125014
TI Transcriptional induction of the ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** in brain metastases of murine K-1735 melanoma
AU Radinsky, Robert (1), Beltran, Pedro J , Tsan, Rachel, Zhang, Ruodan, Cone, Roger D , Fidler, Isaiah J
CS (1) Dep Cell Biol , Box 173, Univ Texas M D Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030 USA
SO Cancer Research, (1995) Vol 55, No 1, pp 141-148 ISSN 0008-5472
DT Article
LA English
AB Metastatic K-1735 murine melanoma cells are amelanotic in culture or in the subcutis of syngeneic mice. When injected into the internal carotid artery, these cells produce melanotic brain metastases. The production of melanin in ***tumor*** cells growing in the brain was directly correlated with induction of ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** (***MSH*** - ***R***) steady-state mRNA transcripts. K-1735 cells isolated from brain lesions and implanted into the subcutis or grown in culture lose ***MSH*** - ***R*** transcripts and become amelanotic. In contrast to K-1735 cells, B16-BL6 melanoma cells constitutively produce melanin and express high levels of ***MSH*** - ***R*** mRNA regardless of the site of growth. Somatic cell hybrids between K-1735 and B16 cells produced melanin and expressed high levels of ***MSH*** - ***R*** mRNA transcripts, regardless of the site of growth, suggesting the dominance of the B16 phenotype. Treatment with alpha-MSH failed to up-regulate ***MSH*** - ***R*** expression in cultured K-1735 cells or to maintain ***MSH*** - ***R*** expression in K-1735 cells isolated from brain metastases to be grown in culture. Responsiveness to alpha-MSH as determined by cell proliferation, melanin production, and intracellular accumulation of cyclic AMP directly correlated with ***MSH*** - ***R*** expression. These data demonstrate that a specific organ environment influences the phenotype of metastatic cells by regulation of specific genes that encode for cell surface receptors.

L3 ANSWER 21 OF 25 CAPLUS COPYRIGHT 2002 ACS
AN 1996:160489 CAPLUS
DN 124:225122
TI UVB-induced melanogenesis involvement of POMC-derived .alpha.-MSH and ACTH
AU Funasaka, Yoko; Chakraborty, Ashok K ; Ohashi, Akiko, Nagahama, Michiko; Ichihashi, Masamitsu
CS School Medicine, Kobe University, Kobe, Japan
SO Photomed Photobiol (1995), 17, 73-6 CODEN: PHPHEA, ISSN: 0912-232X
DT Journal
LA English
AB We asked whether .alpha.-MSH/ACTH/MSH receptor system is involved in UV B (UVB)-induced melanogenesis using cultured normal human keratinocytes and melanocytes. We also examd the effect of UVB-induced cytokines (IL-1 .alpha., .beta., TNF-.alpha.) and quenchers of H2O2 (N-acetylcysteine, NAC) on this system. To evaluate .alpha.-MSH/ACTH prodn and secretion, we performed RIA. The MSH receptor regulation was studied by the binding assay using 125I labeled Nle4DPhe7 alpha.-MSH and by Northern blotting using melanocortin 1 receptor (MC1-R) cDNA as a probe. UVB upregulated .alpha.-MSH/ACTH prodn and secretion of keratinocytes but this activation was inhibited by NAC, meanwhile UVB upregulated prodn. but not secretion of .alpha.-MSH/ACTH in melanocytes. UVB stimulated .alpha.-MSH receptor binding activity of melanocytes with increased MC1-R expression. IL-1 and ET-1 upregulated ***MSH*** - ***R*** binding activity but TNF-.alpha. inhibited it. These results suggest that UVB upregulates both the secretion of MSH/ACTH of keratinocytes and .alpha.-MSH receptor binding activity of melanocytes, resulting in synergistic activation of MSH/ACTH/ ***MSH*** - ***R*** system in a paracrine manner leading to UVB-induced melanogenesis in the skin.

L3 ANSWER 22 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1994 108282 BIOSIS
DN PREV199497121282
TI Calmodulin-binding peptides interfere with ***melanocyte*** - ***stimulating*** ***hormone*** ***receptor*** activity and stimulate adenosine 3',5'-monophosphate production in M2R mouse melanoma cells.
AU Eshel, Yoav, Salomon, Yoram (1)
CS (1) Dep Hormone Research, Weizmann Inst Sci , Rehovot 76100 Israel
SO Endocrinology, (1994) Vol 134, No 1, pp 177-185 ISSN 0013-7227
DT Article
LA English
AB The MSH receptor belongs to a unique class of G-protein-coupled receptors, in which calcium ions control the binding affinity of MSH by a yet unknown mechanism. Possible involvement of a calciumbinding protein (e.g. calmodulin (CaM)) in the regulation of MSH receptor activity has been studied in the M2R mouse melanoma cell line. In this study, we tested the inhibitory effects of a group of calmodulin-binding peptides (CBPs) on MSH receptor activities in intact M2R cells and membrane preparations derived from them. We also report here on stimulatory effects of CBPs on cAMP production in M2R cells that could not be produced in other cell lines lacking MSH receptors. This group of CBPs includes synthetic peptides comprising the CaM-binding domains of Ca-2+/CaM-dependent enzymes, cytotoxic venom peptides, and peptide hormones that have been reported to

directly interact with CaM. The results show that CBPs, at micromolar concentrations, inhibit MSH binding and consequent adenylate cyclase stimulation in a specific and concentration-dependent manner, but have no effect on adenylate cyclase stimulation by prostaglandin E-1. On the other hand, when MSH was omitted and forskolin (0.5-1 mu-M) was added instead, CBPs had the opposite effect on cAMP production, stimulating it in M2R cells, but not in other cell types tested. Thus, these peptides can be considered as antagonists of MSH receptor and partial agonists of M2R adenylate cyclase. In contrast to MSH, the stimulatory effects of CBPs were unaffected by EGTA, suggesting a Ca-2+-independent action of these peptides. Using phospholipid vesicles and M2R cells, we recently showed that CBP activity in M2R cells may include direct partition into the lipid bilayer of the cell membrane, permitting interaction with hydrophobic lipid-inserted domains of components of the signal transducing machinery. Based on these findings, we suggest that the mechanism of action of CBPs in the M2R cells includes two major components: 1) interaction with the cell surface membrane and penetration into the lipid milieu, and 2) interaction with exposed or lipid-embedded protein epitopes intrinsically associated with the MSH-receptor system, thereby affecting the MSH receptor cascade.

L3 ANSWER 23 OF 25 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1994:288152 BIOSIS
DN PREV199497301152
TI Transcriptional induction of the ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** alpha- ***MSH*** - ***R*** in K1735 metastatic murine melanoma cells growing in the brain of syngeneic mice.
AU Radinsky, R. (1), Tsan, R.; Petty, C. M.; Zhang, R., Price, J. E.; Cone, R.; Fidler, I. J.
CS (1) Dep. Cell Biol., U.T.M.D. Anderson Cancer Cent., Houston, TX 77030 USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1994) Vol. 35, No. 0, pp. 62.
Meeting Info.: 85th Annual Meeting of the American Association for Cancer Research San Francisco, California, USA April 10-13, 1994
ISSN: 0197-016X.
DT Conference
LA English

L3 ANSWER 24 OF 25 CAPLUS COPYRIGHT 2002 ACS
AN 1994:125072 CAPLUS
DN 120:125072
TI The melanocyte-stimulating hormone (MSH) receptor in M2R mouse melanoma ***tumors*** solubilization and properties of the receptor-MSH complex and its covalently crosslinked conjugate
AU Shafir, I.; Schmidt-Sole, J.; Shai, E.; Salomon, Y.
CS Dep. Horm. Res., Weizmann Inst. Sci., Rehovot, 76100, Israel
SO Melanoma Res. (1993), 3(3), 157-68
CODEN: MREEEH; ISSN: 0960-8931
DT Journal
LA English
AB Several properties of the MSH receptor in solid melanotic and amelanotic mouse M2R ***tumor*** isografts were studied in C57BL mice. Using cell membrane fractions prepd. from such ***tumors*** and the superpotent [Nle4,D-Phe7] alpha MSH analog, the affinity and receptor contents of the two ***tumor*** variants were similar. When occupied by MSH, the receptor-MSH complex (R.cntdot.MSH) was readily sol. in cholate. In the solubilized form, R.cntdot.MSH was extremely stable and dissocd. to an extent of only 30% within 12 days at 4.degree.. While this high stability can be maintained in the pH range of 7.0-8.5, the solubilized R.cntdot.MSH complex becomes increasingly unstable below pH 7.0 and totally dissocs. at a pH <6.0. In the membrane-bound form, the R.cntdot.MSH complex shows a parallel pH stability profile which is shifted down by approx. two pH units. In addn. to low pH, the R.cntdot.MSH complex becomes unstable and totally dissocs. in the presence of 10 mM EGTA, suggesting that the calcium-sensitive function of the receptor is still assocd. with the receptor in the detergent-sol. state. The R.cntdot.MSH complexes in the sol. and membrane-bound forms are also totally resistant to proteolytic digestion by V8 protease, but were slowly digested by trypsin. Treatment of R.cntdot.MSH with 1-ethyl-3-(3-dimethylamino-propyl)carbodiimide hydrochloride or bis(sulfosuccinimidyl) suberate led to covalent crosslinking of MSH to the receptor mol. The electrophoretic mobility on SDS-PAGE of the 43/46 kDa doublet of the receptor-MSH conjugate (R*MSH) was identical to the photoaffinity labeled MSH receptor product described earlier in cultured M2R cells. However, the efficiency of prodn. of the crosslinked product was .apprx. 30%, much higher than that achieved previously by photoaffinity labeling. Using rabbit polyclonal anti-alpha MSH antibodies, the R*MSH conjugate was identifiable on Western immunoblots. These results provide a basis for further development of procedures for purifn. of the MSH receptor mol. and studying its protein structure.

L3 ANSWER 25 OF 25 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 75026565 EMBASE
DN 1975026565
TI Regulation of melanocyte stimulating hormone action at the receptor level: discontinuous binding of hormone to synchronized mouse melanoma cells during the cell cycle.
AU Varga J.M.; Dipasquale A.; Pawelek J.; et al.
CS Dept. Dermatol., Yale Univ. Sch. Med., New Haven, Conn. 06510, United States
SO Proceedings of the National Academy of Sciences of the United States of America, (1974) 71/5 (1590-1593).
CODEN: PNASA6
DT Journal
FS 037 Drug Literature Index
016 Cancer
003 Endocrinology
029 Clinical Biochemistry
LA English
AB Melanocyte stimulating hormone coupled to Sepharose effects an increase in tyrosinase (EC 1.14.18.1, monophenol monooxygenase) activity of cultivated mouse melanoma cells. Synchronized cells are found to respond to melanocyte stimulating hormone only in the G2 phase of the cell cycle, although their response to adenosine 3',5' cyclic monophosphate (cAMP) is independent of position in the cell cycle. The binding of 125I-labeled melanocyte stimulating hormone occurs predominantly in G2. These observations are satisfied by a model in which the hormone can activate adenylate cyclase (EC 4.6.1.1) by binding to a ***melanocyte*** ***stimulating*** ***hormone*** ***receptor*** only in G2, the events distal to cyclic AMP production can occur throughout the cell cycle.

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---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	SESSION	TOTAL
FULL ESTIMATED COST		97.87	98.02

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	SESSION	TOTAL
CA SUBSCRIBER PRICE		-1.86	-1.86

STN INTERNATIONAL LOGOFF AT 14:39:41 ON 11 MAR 2002

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